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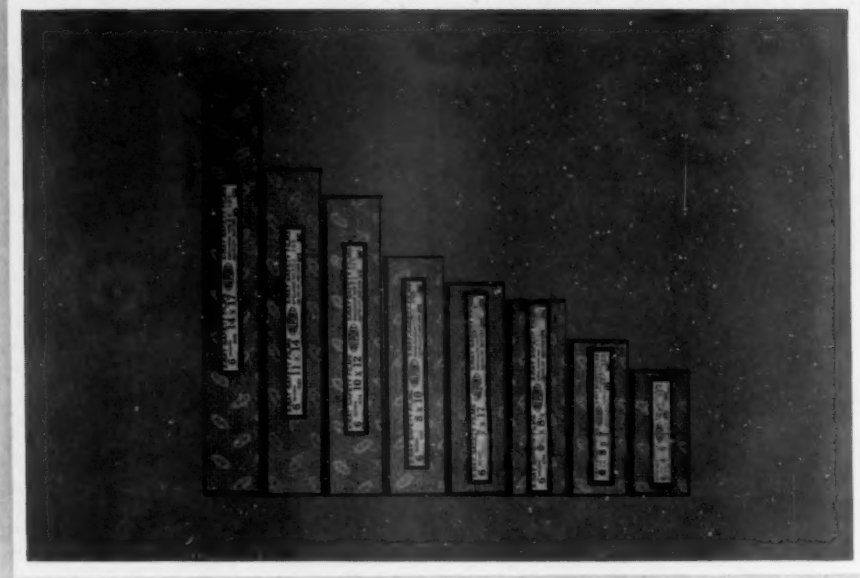

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VOL. 45 APRIL, 1941 No. 4

THE TUBERCULIN TEST AND ROENTGENOGRAM IN THE EARLY TUBERCULOUS LESION*

By THEODORE L. BADGER, M.D., *Chief of the Thoracic Clinic,*
and MAX RITVO, M.D., *Visiting Roentgenologist*
Boston City Hospital
BOSTON, MASSACHUSETTS

THE small early tuberculous lesion has assumed a new significance in recent years. Formerly the "spot on the lung" was a term used glibly and naively by physicians to assure the patient that his tuberculous infection had left behind no more than a small scar which he could safely forget. Today we know that the potentialities of that "spot" are out of all proportion to its size. We know, too, that the percentage of correct diagnoses of minimal tuberculosis is exceedingly low. Eighty-seven per cent of tuberculous disease is not discovered until it has passed beyond the minimal stage.[4] The responsibility for this poor showing in early diagnosis falls heavily on the family physician and the internist, but the roentgenologist has no small share in evaluating the early tuberculous lesion.

Johannes Heimbeck,[5] a Norwegian internist, in 1928 first published the result of his studies of tuberculosis in nurses at the Ullevaal Municipal Hospital in Oslo. He tuberculin tested all nurses entering the training school and found that 50 per cent of them failed to react to tuberculin. In other words, one-half of his nurses entering training had never been exposed to tuberculosis and were ready for their primary or childhood type of infection in adult life. These findings were contrary to accepted belief at that time, namely that most people had been infected with the tubercle bacillus by the age of twenty. Heimbeck found, by retesting his nurses with tuberculin, that all but a few of the negative reactors turned positive during their three year training. During their student days the exposure to tuberculosis was essentially the same for all nurses, and the living conditions and hours of work were similar within the hospital. In this well controlled clinical study, he found tuberculosis four times as frequent in those who started training tuberculin-negative as in those tuberculin-positive. Heimbeck has repeatedly called attention to the very high morbidity in this tuberculin-negative group.

Subsequent surveys in hospitals and schools in America brought somewhat similar but less striking results. Amberson and Riggins[2] at Bellevue Hospital in New York also found approximately half their nurses negative reactors on admission to training.

* From the Thorndike Memorial Laboratory, Second and Fourth Medical Services (Harvard), the X-Ray Department, Boston City Hospital, and the Department of Medicine, Harvard Medical School. Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

All but a few of these turned positive during three years of study on the hospital wards. Two to three times as much tuberculosis appeared in those initially tuberculin-negative. Shipman and Davis,[9] on the contrary, at the University of California Hospital, state that the majority of their

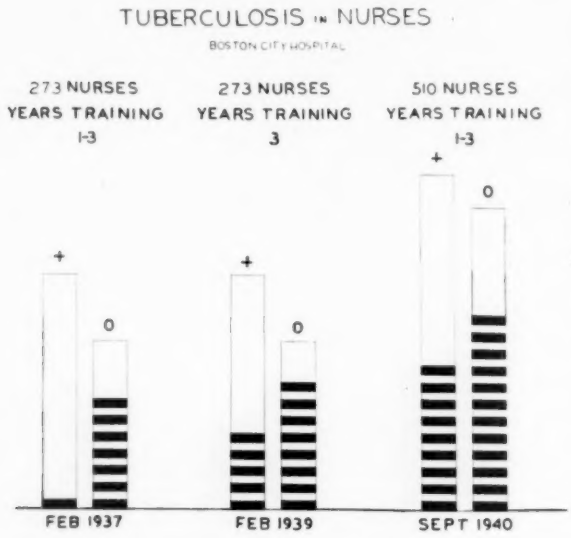

Chart I. Distribution of tuberculous lesions among those nurses positive to tuberculin on admission to training and those negative to tuberculin who became positive. (Each bar represents one case.)

negative reactors remained so through three years of training. They, however, had few cases of tuberculosis on the wards and exposure was slight.

Myers, Diehl and Lees[8] have called attention to the increase of infection in schools of nursing with and without tuberculosis services as compared with schools of education. This is well shown in Table 1.

At the Boston City Hospital a survey of tuberculosis was begun in 1932. A tuberculin test, stereoscopic roentgenograms of the lungs, and a complete physical examination were required of all nurses admitted to training. Retesting of all negative reactors was carried out every six to twelve months. Stereoscopic roentgenograms of the lungs were taken yearly irrespective of the tuberculin test and every six months on negative reactors who turned positive. Fifty-seven per cent of our nurses were positive to tuberculin when admitted to training and 95 per cent were positive at graduation. Badger and Spink[3] reported the results of this study in 1937.

Chart 1 shows the distribution of roentgenographic lesions in those nurses initially positive and negative to tuberculin. We see that in 1937 the 273 nurses who had completed one to three years of training showed 7 roentgen lesions in the initially negative reactors as compared with one lesion in those who started training tuberculin-positive. In 1939 when all members of the same group had completed their three years of training, the distribution of cases in the two groups had changed. In 1940 the total of 510 nurses includes the 273 who had completed three years of training in 1939 and has added to it another 237 nurses in training one to three years. In 1940 there are 12 roentgen lesions in the initially negative reactors and 9 lesions in the initially positive reactors. It is of interest that the number of roentgen lesions appearing in these nurses becomes more equally distributed between the two groups of reactors, with the passage of time. In our experience the differentiation of positive reactors at the start of training and the negative reactors who later become positive appears to be a relatively unimportant factor.

If we now analyze the morbidity of these two groups of initially positive and negative reactors and correlate the clinical characteristics of each with its roentgen

Table I[8]

Institution	Percentage of Positive Tuberculins
Hospital with tuberculosis service	
Probationers	39
Seniors	100
Hospital without tuberculosis service	
Probationers	28
Seniors	48
College of Education	
Freshmen	23
Seniors	28

lesion, certain facts will become evident. Heimbeck[6] in a 1938 report of his studies speaks of benign and malignant forms of tuberculosis in his nurses but classifies them all as "diseased." In our studies at the Boston City Hospital we have observed that some of these small early lesions have been so benign as to clear spontaneously or leave behind only a small healed scar while the nurses have remained in training. Other lesions have required the usual treatment for active tuberculosis. Chart II analyzes the character of the roentgen lesions in the two groups of initially positive and negative reactors. Here we have classified as "clinical tuberculosis" all cases requiring treatment for the cure of their disease. We have classed as "x-ray tuberculosis" those whose process was so benign as to heal spontaneously without ever having shown clinical signs or symptoms of activity.

In Chart II we see that in those positive to tuberculin on entrance 7 of the 9 tuberculous lesions required sanatorium care. Two, however, who had lesions visible by the roentgenogram healed spontaneously and never presented signs or symptoms of clinical disease.

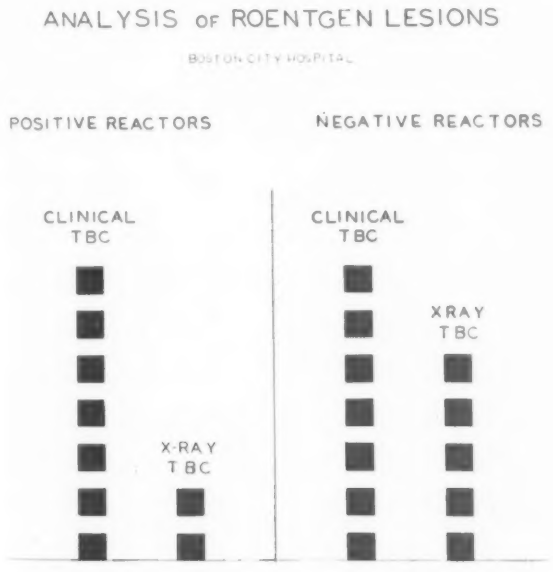

Chart II. An analysis of the tuberculous lesions which appeared in those nurses who entered training positive to tuberculin, and those negative to tuberculin who became positive. (Each square represents one case.)

Fig. 1. Case 1. June 14, 1933. Primary tuberculosis. Small parenchymal lesion just below the anterior end of the right third rib, with greatly enlarged hilar gland. This was followed in a month by acute pleurisy with effusion on the right.

In the initially negative reactors, 7 of the 12 tuberculous lesions required sanatorium care and 5 presented lesions that were so benign as to heal or entirely disappear while the nurses remained on duty. The 7 clinical cases of tuberculosis in each group which needed treatment required essentially the same therapy to cure their disease. From an analysis of these lesions it becomes apparent that no sweeping conclusions can be drawn regarding the vulnerability of those initially tuberculin-negative as compared with those tuberculin-positive.

REPORT OF CASES

A few characteristic roentgen lesions from these groups are shown with a brief clinical history.

Case 1. J. F., a girl, aged eighteen, of French parentage who entered training in February, 1932. Roentgen examination of the lungs and the tuberculin reaction were negative. Six

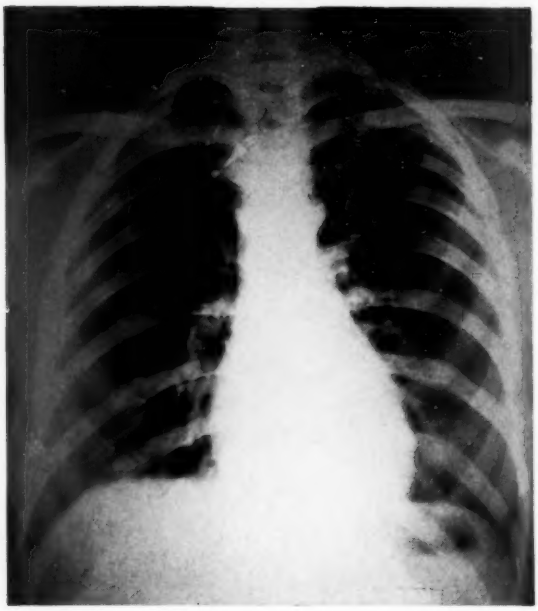

Fig. 2. Case II. August 7, 1934. Small area of infiltration between the second and third anterior ribs in the periphery of the left chest. In May, 1935, she developed an acute pneumonic consolidation at left apex in spite of prolonged hospitalization. Sputum negative.

months after admission to the hospital, the tuberculin test became positive. A year after admission, a routine roentgenogram of the lungs showed a small parenchymal infiltration in the right mid-chest with a greatly enlarged right hilar lymph node (Fig. 1). Physical examination at that time was negative and the only symptom was a slight pleuritic pain in the right axilla of three days' duration. Rest treatment at home was prescribed. The fifth month after the discovery of the lesion, a pleurisy with effusion followed an acute upper respiratory infection. This girl presented a characteristic childhood type of tuberculosis in an adult, followed by a pleurisy with effusion. Usually these cases come to our attention because of the pleurisy with effusion without roentgen evidence of the primary infection.

Case II. M. C., a Scotch girl, aged eighteen, commenced training in September, 1932, with a negative physical examination, a negative tuberculin test and a negative roentgenogram of the lungs. In July, 1934, she developed a low-grade fever and was found to have a positive tuberculin test for the first time. A roentgenogram of the lungs revealed a very small shadow in the periphery of the left second anterior space (Fig. 2). Joint pains in the third week, however, led to the diagnosis of rheumatic fever as the cause of her fever. She was treated with bed rest for three months in the hospital and then sent home to continue her bed rest and convalescent care. In February, 1935, a check-up revealed no further activity in her rheumatic fever and the roentgen picture of the lungs was unchanged. She was allowed to return to work but after two weeks she fainted on the hospital ward. Because of some recurrence of pain in the joints, she was still thought to have rheumatic fever. She failed, however, to respond to therapy and her fever rose slowly over a period of weeks. Physical examination of the lungs was repeatedly negative but roentgen study of the chest in May, 1935, revealed a pneumonic infiltration in the left upper lobe, proven to be tuberculous.

Case III. M. R., an Irish girl, aged eighteen, began training in September, 1934, negative in all respects. In May, 1935, she was admitted to the hospital for a severe respiratory infection. At that time she still had a negative tuberculin test and a negative roentgenogram of the lungs. September 8, 1935, three months later, she was admitted to the hospital with symptoms of cys-

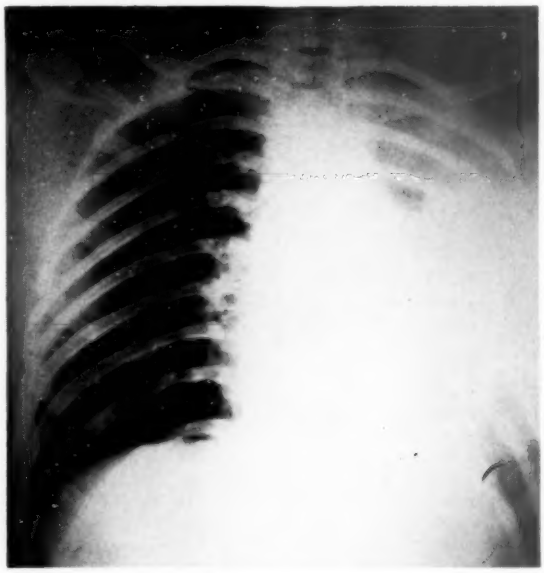

Fig. 3. Case III. September 19, 1935. Acute pneumonic consolidation of entire left upper lobe. Sputum contained great number of tubercle bacilli. Tuberculin test positive. Three months previously roentgenogram of the lungs negative and tuberculin test negative.

titis following another upper respiratory infection. Physical examination was negative but a roentgenogram of the lungs showed a delicate fan-like infiltration extending into the apex and infraclavicular region of the left lung. Three days later signs of consolidation appeared in the left axilla together with a positive sputum. Ten days after the onset of her illness, there was massive consolidation of the left upper lobe (Fig. 3).

Case iv. G. McD., an American girl, aged eighteen, entered training September, 1932, with a negative tuberculin test, a negative roentgenogram of the lungs and negative physical examination. In January, 1934, a routine roentgen examination of the lungs revealed a small, soft infiltration at the left apex (Fig. 4). Because of her sense of well being and complete freedom from symptoms and signs the nurse requested that she be allowed to remain on duty. This was permitted against our better judgment. Monthly roentgenograms with clinical and laboratory check-ups showed a progressive diminution in the size of the original lesion. Six years later, she was well and working with only a small scar demonstrable.

Case v. M. C., an American girl, aged eighteen, began training in September, 1934, with a negative tuberculin test, a negative roentgenogram of the lungs and a negative physical examination. Five months later, her tuberculin test had become positive and the roentgenograms of the lungs showed a small homogeneous shadow in the periphery of the right first interspace (Fig. 5). Owing to her well being and complete freedom from symptoms and signs of disease, she also was allowed to remain in training with a monthly roentgen examination of the lungs and clinical observation. Her course was one of uneventful clearing of the roentgen lesion during the ensuing eight months. Four and one-half years after the disappearance of this lesion, she is known to be well without further abnormal shadows in her lungs.

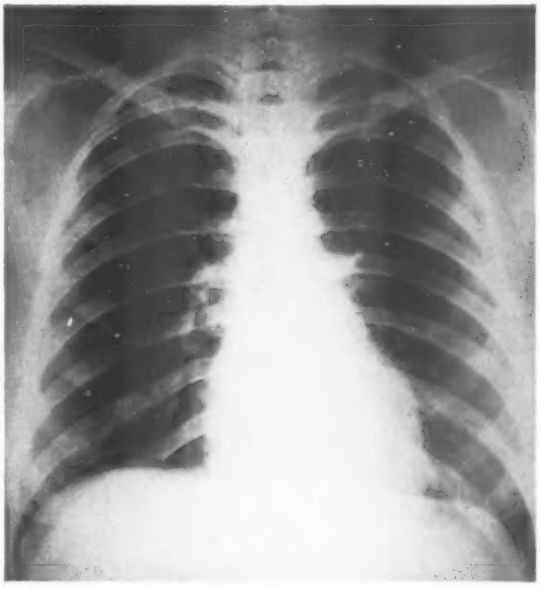

Fig. 4. Case iv. January 12, 1934. Small homogeneous density in apex of left lung. Progressive healing of the lesion while the nurse remained on duty. Small scar now remains.

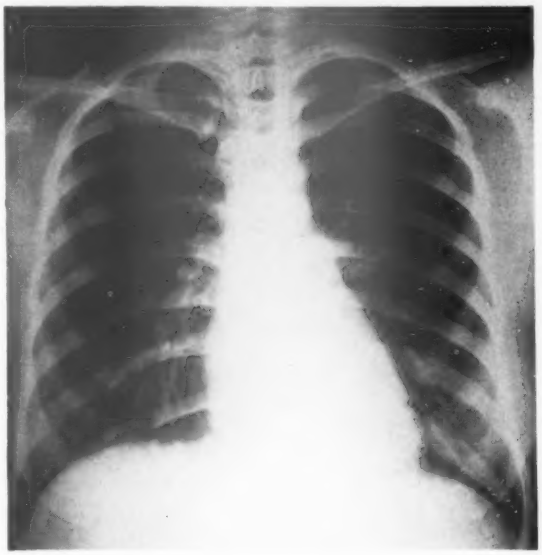

Fig. 5. Case v. February 12, 1935. Small homogeneous infiltration in the periphery of the right lung between the first and second interspace anteriorly. This cleared entirely in eight months while the nurse remained on duty.

Case vi. E. P., a student nurse, aged eighteen, who entered training in September, 1938, with a positive tuberculin test and a negative roentgenogram of the lungs. She remained well but routine roentgen examination of the lungs in January, 1940, showed a soft flocculent infiltration on the right extending from the hilum into the right mid-chest (Fig. 6). She was symptom-free and physical examination was negative at the time of the roentgenogram. Hospitalization was immediately instituted. A lateral roentgenogram taken later, after the institution

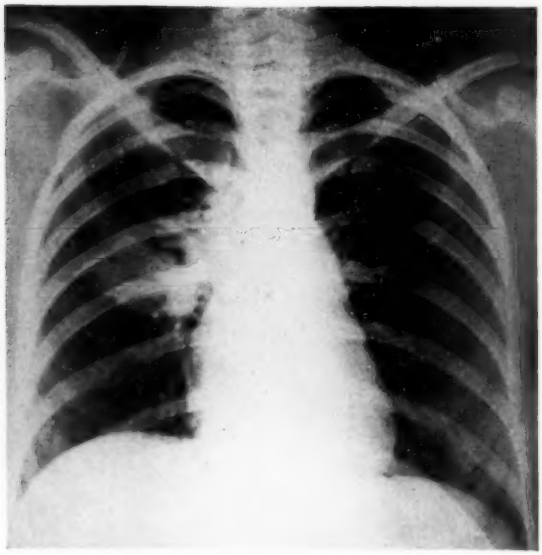

Fig. 6. Case vi. Infiltration in right mid-chest extending out from the hilum found on routine roentgen examination of the lungs. Lateral view showed this lesion to be in the apex of the right lower lobe.

of pneumothorax, showed the lesion to be in the apex of the right lower lobe.

Evaluation of the Tuberculin Test

The tuberculin test should be used as a means of detecting the presence of tuberculous infection. A negative tuberculin test in strong healthy young people indicates the absence of tuberculous infection. But no tuberculin test can be considered negative unless the individual has failed to respond to 1 mg. of old tuberculin or its equivalent.

A positive tuberculin test proves the presence of infection somewhere in the body. But a positive test does not afford any information concerning the anatomical size of the lesion, or its degree of activity or inactivity in a given case.

A negative test that becomes positive indicates the appearance of a so-called primary or first-infection focus of tuberculosis. At the Boston City Hospital we have shown that practically all of those tuberculin-negative at the beginning of training later become positive, but only a few of these develop roentgenologic evidence of infection. From this we, as others, have concluded that the primary infection in adults *may* be as benign a process as it usually is in children.

In those nurses initially positive to tuberculin on entering training, we have no knowledge as to when the primary infection occurred or when the tuberculin test became positive. It is logical, therefore, to assume that at the time they began training a certain number reacting positively had only recently become infected. Their status then is closely similar to those entering training with a negative tuberculin test who become exposed to tuberculosis shortly after admission and then develop a positive tuberculin test. The majority of both these groups develop the usual benign primary infection in a region not demonstrable by roentgen examination. A few present small but definite roentgen lesions without clinical manifestation of disease. Others progress to clinically active tuberculosis.

It is illogical to believe that any strict line can be drawn between those who begin training tuberculin-positive and those who change from negative to positive after admission to training. Time appears to be the vital factor in demonstrating which individuals will develop immunity or constitutional resistance to the tubercle bacillus following this primary infection, and which will develop destructive disease. Myers, Ch'iu and Streukens[7] believe that *all* primary infections in adults as well as in children are benign and require no treatment. This has not been our experience, as we have shown above.

Evaluation of the Small Early Tuberculous Lesion

It becomes evident from an appraisal of these cases that it is impossible to anticipate from a single observation of the roentgenogram what the small early tuberculous lesion will do in any given individual. Complete clinical and laboratory studies should accompany the observation of each case. Knowledge of a family history of tuberculosis may be important. Known contact

with tuberculosis through occupational hazards or the care of the tuberculous sick may contribute to the evaluation of the lesion. Symptoms suggestive of active disease may also be present. Sputum if obtainable may be positive, or examination of the fasting gastric contents may show tubercle bacilli. But the small roentgen lesion may be the single positive finding when a study of the case has been completed.

Definite diagnosis of this small lesion as tuberculosis, in the presence of a positive tuberculin test, hinges upon its persistence in the roentgenogram. Bronchopneumonia, patches of atelectasis and small areas of infarction will usually disappear promptly. Early bronchogenic carcinoma originating in the terminal bronchioles may produce shadows indistinguishable from the early tuberculous lesion. Once the diagnosis of tuberculosis has been made, however, a careful correlation of all facts is necessary to determine the character of the process. It must, if possible, be decided whether the lesion is an old healed scar, a latent process which may break down at some future time, an early destructive lesion, or a benign and innocent process which will heal spontaneously. Repeated roentgenological studies, weekly and then monthly, may be necessary before the true character of the lesion can be determined. Amberson[1] has expressed his belief that the latent quality of these small early tuberculous lesions requires bed rest to effect a lasting cure. We are in accord with this principle but believe that a few cases may go on to spontaneous healing. Constitutional resistance to tuberculosis is a feature of the disease which cannot be predicted, but it is a most important aspect in prognosis and in the development of immunity to the tubercle bacillus. Young women in their late teens and early twenties have less constitutional resistance to tuberculosis than males of a similar age group. Advancing age appears to bring increasing resistance to the disease. The frequency of exposure to the tubercle bacillus and the size of the pulmonary lesion bear a direct relation to the progress of the disease. Our experience at the Boston City Hospital has led us to the belief that tuberculosis is less closely associated with the acquisition of allergy as shown by the development of a positive tuberculin test, than with age, sex, race, occupation and severity of contact. Wherever there is careless exposure to the tuberculous sick, there will occur the highest incidence of infection and active disease. With the increasing use of roentgenologic surveys, it becomes a growing responsibility of the roentgenologist to recognize the small tuberculous lesion and to use sound judgment in interpreting its character. Its latent qualities demand careful supervision and in our opinion this can best be done by the close cooperation of the internist and the roentgenologist.

SUMMARY

1. A study of tuberculosis in nurses at the Boston City Hospital, carried on since 1932, has shown that the morbidity is almost equally distributed between those who enter training tuberculin-positive and those who enter tuberculin-negative.

2. All but a few of the nurses who enter training with a negative tuberculin test become positive during their three years on the wards. The majority of those who turn positive develop the usual benign primary infection in a region not demonstrable by the roentgenogram. A few develop small but definite roentgen lesions without clinical manifestation of active disease. Others progress to clinically active tuberculosis.

3. Having established the diagnosis of the early tuberculous lesion, by its persistence in the roentgenogram, it must, if possible, be determined whether it is an old healed scar, a latent process potentially active in the future, an early destructive infiltration or a benign lesion which will heal spontaneously.

4. It becomes a growing responsibility of the roentgenologist to recognize these small tuberculous infiltrations. He must understand their latent potentialities which

we have shown, and in cooperation with the internist, use sound judgment in interpreting their characteristics.*

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* For discussion see pages 492 and 517.

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TUBERCULOSIS OF THE MEDIASTINUM*

By CHESTER S. KEEFER, M.D.
BOSTON, MASSACHUSETTS

TUBERCULOSIS of the mediastinum usually includes (1) enlargement of the tracheobronchial lymph nodes, (2) tuberculous abscess of the anterior or posterior mediastinal space, (3) chronic fibrosis of the lymph nodes with and without calcification, and an associated fibrous mediastinitis resulting from a healing or healed tuberculosis.

For purposes of description and diagnosis, it is well to divide the mediastinal space into the anterior and posterior mediastinum, the former being the space anterior to the trachea and the latter the space posterior to it. This division is helpful since the symptoms and signs produced by a process in the anterior mediastinum are different from those in the posterior mediastinum, and the roentgen examination should be made with these two divisions in mind.

The diagnosis of tuberculous mediastinitis depends on: (1) the symptoms and signs of an infection, (2) the symptoms and signs by physical and roentgen examination of the chest of a process in the mediastinum which is causing compression signs, and (3) the associated signs of tuberculosis.

SYMPTOMS AND SIGNS OF INFECTION

There is nothing distinctive or characteristic about the symptoms or signs of infection in this disease. Fever, loss of weight, and other constitutional symptoms of infection are common. A most careful physical examination may fail to reveal the localizing signs suggesting the cause of the symptoms.

SYMPTOMS AND SIGNS BY PHYSICAL AND ROENTGEN EXAMINATION OF THE CHEST

Of these two examinations, the roentgen examination is by far the more important so far as revealing the cause of the illness is concerned. When physical signs are present and the process is in the anterior mediastinum, an increase in the retromanubrial dullness and the signs of compression of the innominate vein or superior vena cava are the most important. Occasionally, if the process has extended beyond the mediastinal lymph nodes, there may be actual local bulging of the chest wall at the interspaces on either side of the sternum and, in rare cases, a sinus may form.

When the process is localized in the posterior mediastinum, the important symptoms are difficulty in swallowing and cough. These symptoms are due to compression of the esophagus and irritation of the trachea. Physical signs may reveal dullness and loud whispered spoken voice sounds over the upper part of the spine.

ROENTGENOLOGIC DIAGNOSIS OF MEDIASTINITIS

An excellent discussion of the roentgen diagnosis of tuberculous mediastinitis has been presented by Kornblum and Osmond,[5] who stress the following points: (1) widening of the mediastinal shadow with a unilateral or bilateral bulging of the mass, which is smooth, regular, and not nodular; (2) deviation of the trachea to the side of greater involvement. When there is calcification of the enlarged lymph nodes, then the diagnosis is somewhat simplified. There may be no signs of pulmonary involvement in any of these cases.

When tuberculosis of the mediastinum is suspected without any evidence of a parenchymal process in the lungs, then the spine should be investigated for signs of Pott's disease.

Milles[6] quotes McPhedran and Kornblum and Cooper as saying that the roentgen findings in mediastinal tuberculosis

* From the Evans Memorial, Massachusetts Memorial Hospitals, and the Department of Medicine, Boston University School of Medicine, Boston. Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

may be negative or misleading unless the glandular enlargement is marginal and discrete or the seat of calcium infiltrations. My own observations concerning the roentgen signs of tuberculosis of the mediastinum are included under the discussion of tuberculosis of various parts of the mediastinum.

ASSOCIATED FEATURES

The nature of the process in the mediastinum is frequently suggested by the associated lesions in the lung, spine, skin, pericardium, or the superficial lymph nodes. When there are signs of infiltration in the lungs and tubercle bacilli in the sputum, the diagnosis of the nature of a mediastinal process is always easier than when there is no infiltration in the lungs. Destruction of one or more vertebrae with the signs of an abscess in the mediastinum is excellent evidence for tuberculosis although somewhat the same picture may be produced by *Staphylococcus aureus* osteomyelitis with mediastinal abscess. The presence of erythema nodosum or tuberculids in the skin and enlargement of the tracheobronchial lymph nodes is always suggestive that the process is due to tuberculosis.

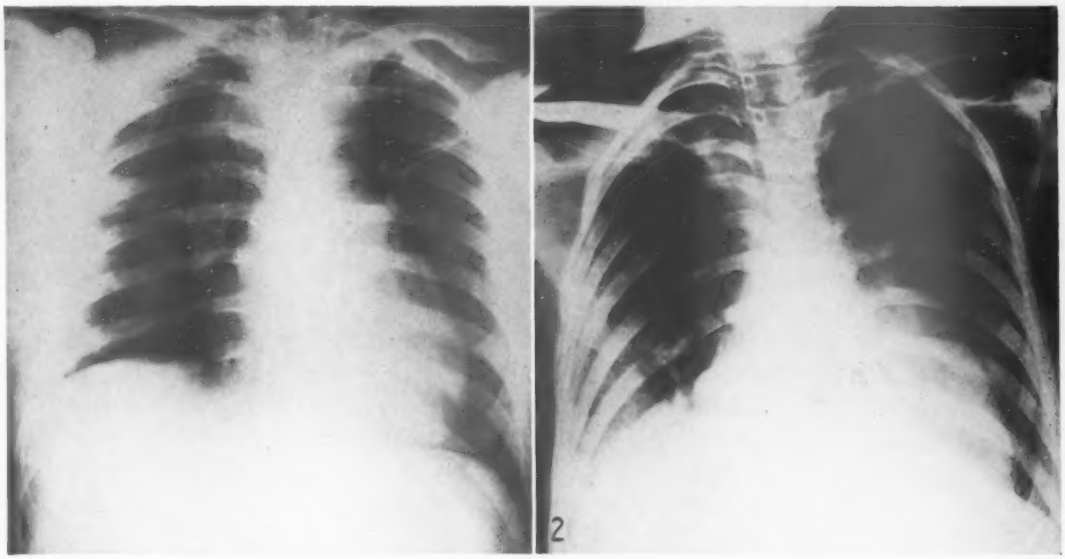
Milles explains mediastinal tuberculosis in adults without demonstrable lesions in the lungs as an activation of tuberculous infection in the tracheobronchial lymph nodes. He also records his observations of tuberculosis of the mediastinum associated with a cold abscess and a toxic nephrosis with tuberculosis of the pericardium or miliary tuberculosis.

ENLARGED BRONCHIAL AND MEDIASTINAL LYMPH NODES

This is the commonest form of tuberculosis of the mediastinum and it is more frequent in children than in adults. The diagnosis is most often made by the roentgenologist. It scarcely need be said that the lesions in the lung in children may be small and the glands may reach considerable size. This is characteristic for so-called childhood tuberculosis. In adults it is infrequent to observe massive enlargement of the lymph nodes of the mediastinum with minimal lesions in the lungs, but that such cases occur there is no doubt. They have been called intrathoracic tuberculous lymphoma.[1,7] Wessler[7] described 5 cases in 1928 and directed attention to the following features. All of the cases studied by him occurred in young adult women. There were no pathologic changes in the lungs in 4, and the diagnosis was made by means of roentgen examination of the chest and by excision of the enlarged supraclavicular lymph nodes and finding miliary tubercles in them. The constitutional symptoms of a chronic infection were present and no change in the size of the nodes was detected over a period of six months to three years. In the same year, Buckley[1] made a brief report of a patient who had a large mass of tuberculous lymph nodes in the upper mediastinum which had compressed the trachea and arch of the aorta. Death resulted from a massive pulmonary hemorrhage.

It is quite possible that many of the cases of enlarged lymph nodes of the mediastinum which have been labeled tuberculous have been instances of sarcoid, and this is especially true of the cases in which the glands show the histologic picture of miliary tuberculosis without the finding of tubercle bacilli. Other distinguishing features in favor of sarcoid may be the presence of chronic fibrotic changes in the lungs, cystic changes in the bones of the hands and feet, a negative tuberculin reaction, hyperproteinemia, and the tendency to spontaneous regression of the process.

In a previous paper,[4] in which I discussed the causes of obscure fever, I reported a case of tuberculosis of the lymph nodes of the mediastinum and supraclavicular region in which the diagnosis was established only after a lymph node above the clavicle had been excised and examined histologically.



Figs. 1 and 2. Tuberculosis of the posterior mediastinum. The process began in the upper third of the posterior mediastinum (Fig. 1) and finally spread downward (Fig. 2) to involve the entire right side of the mediastinum. The patient had high, irregular fever for one year, erythema nodosum, and arthritis which proved to be tuberculous in origin.

TUBERCULOSIS OF THE ANTERIOR MEDIASTINUM

Tuberculosis of the anterior mediastinum is due to suppuration of lymph nodes or extension of infection to the mediastinum from the sternum. It may produce enlargement of the lymph nodes, an abscess, or a chronic fibrotic process which constricts various structures. An abscess in this area may perforate the chest wall forming a sinus in the neighborhood of the sternum. A chronic fibrotic process may cause signs of compression of the superior vena cava or innominate veins and, in some cases, it is accompanied by signs of a concretio cordis. In my discussion of tuberculosis of the pericardium,[2] I mentioned the importance of tuberculosis of the mediastinal lymph nodes as a common source of infection. The diagnosis of tuberculosis of the anterior mediastinum should be entertained, then, when signs of any of the above processes are present.

TUBERCULOSIS OF THE POSTERIOR MEDIASTINUM

Tuberculosis of the posterior mediastinum arises from the vertebrae (Pott's disease) or from caseous lymph nodes, and the usual result is an abscess. They occur most often in the lower cervical and upper dorsal regions of the thorax, and they commonly form a circumscribed mass that is confused with a solid tumor. Infrequently, a diffuse fibrotic process is found instead of an abscess. These abscesses may gravitate downward from their point of origin and appear in the abdominal wall or in the regions where a psoas abscess perforates.

The symptoms are due to the infection and to the presence of a space-occupying process in the posterior mediastinum. Fever, pain in the back, dysphagia, and gradual wasting are common complaints. The physical signs vary, depending on the size of the abscess and its origin and, indeed, there may be no abnormal physical signs elicited on a most careful examination. When the vertebrae are the source of the abscess, there is tenderness over the spine, but in some cases the focus of necrosis in the spine is so small that this sign will be missing. Decreased resonance and an increase in the whispered voice sounds over the upper part

of the spine may be present and suggest a lesion in the posterior mediastinum.

The most important signs, however, are those revealed roentgenographically. Anteroposterior and lateral views of the chest and spine should be made and the position of the esophagus should be defined. The shadow cast by the abscess may be either in the upper third of the mediastinum or superimposed on the cardiac silhouette. There may or may not be a demonstrable destruction of the vertebrae.

Characteristically, the shadow is homogeneous, with a sharp convex border. Almost invariably it is symmetrical and bilateral. It is situated in front of the spine and behind the esophagus, and its axis is parallel with the long axis of the body. The shadow is seen best in the middle or lower part of the posterior mediastinum; it is superimposed on the cardiac shadow and pushes the esophagus forward (see Figs. 1 and 2).

On a previous occasion, I[3] discussed acute and chronic mediastinitis and described cases of tuberculosis of the posterior mediastinum without involvement of the spine. I should like to say once again that the most important feature in diagnosis is the appearance by roentgen examination.

Chronic diffuse mediastinitis due to tuberculosis is often associated with signs of tuberculosis of the lungs or signs of concretio cordis. When signs of these associated lesions are absent, the commonest group of symptoms and signs are those due to a constriction or thrombosis of the superior vena cava or innominate veins without evidence of a mediastinal tumor or aneurysm of the aorta. In a case of superior vena caval obstruction which was caused by tuberculosis of the tracheobronchial lymph glands and reported by Milles, there was only a slightly widened upper mediastinal shadow which was interpreted as being due to a tumor. At the time of operation no tumor was found and at necropsy a "cartilaginous, rather poorly defined mediastinal mass was found adherent to the lung and encircling the superior vena cava about 4 cm. above the right auricle. When the vessel was opened, its lumen in this region was demonstrated to be 2 mm. in diameter; the wall was thickened to 7 mm. by dense cartilaginous connective tissue, and the center of the encircling mass was made up of cheesy, necrotic material of the shape and appearance of a broken-down lymph node."

SUMMARY

In summing up this discussion, it can be said that tuberculosis of the mediastinum includes tuberculosis of the lymph nodes, cold abscess of the mediastinum secondary to disease of the spine, sternum or lymph nodes, and chronic fibrous mediastinitis.

The diagnosis depends on the symptoms and signs of an infection of the mediastinum, the roentgen examination of the chest, and the anatomical process associated with the mediastinal disease.

The various clinical features of different forms of the disease are discussed.

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DISCUSSION OF PAPERS BY DRS. BADGER AND RITVO, AND KEEFER*

Dr. P. F. Butler, Boston. Regarding Dr. Badger's paper, not much can be added. As a certain number of the early primary lesions are

* For further discussion see page 517.

elsewhere than in the chest, it is obvious that they cannot be discovered by the roentgen ray. Our problem seems to be twofold: first, to pick up these early chest lesions, and second, to try for some evidence that might give us a lead as to whether they will recover quickly or go on to greater involvement. Some data might be obtained by more detailed study of the bronchial trunks near the involved area and also of the associated hilar glands. In lung abscess, for instance, these increased linear markings and connected lymphatic glands are very important signs of a process that is advancing. Serial films at short intervals of a week or two might be helpful in focusing attention over these regions. Information of value might be obtained by comparing spot films of those who recover without any special treatment with those whose pathology is progressive.

As there is considerable controversy over the significance of positive or negative tuberculin tests, it would appear to be the function of roentgenologists to focus their attention on more detailed studies of the chests in question.

In regard to Dr. Keefer's paper, I may say that the roentgen ray is of definite value in mediastinal conditions. The mediastinal region has been thoroughly studied and much accurate information may be obtained. These differential signs are well described in the literature and I will only briefly review them.

Biopsy is a hazardous procedure. The roentgenogram furnishes the most information, but must be correlated with other signs.

We must differentiate between tumors (benign and malignant), substernal goiters and abscesses, thymoma, Hodgkin's disease, enlarged lymph nodes, mediastinal abscess and cyst, and aneurysm.

The following information may be determined by roentgen examination: size, shape, infiltration; single or multiple; location in any particular mediastinum or about the bronchi; bilateral or unilateral; displacement of esophagus or aorta; connection with heart and aorta; pulsation; movement, with diaphragm or ribs. The examination must be both fluoroscopic and with films.

Thymic enlargements are found in children and are radiosensitive.

Substernal thyroid; moves with trachea, no pulsation, may displace trachea. If irregular, fixed, infiltrating and growing rapidly, then cancer is to be suspected.

Benign tumors—cysts, fibroma, lipoma, neurofibroma. They develop slowly, are usually symptomless. Cysts are unilateral, have a fluid level, are round or oval and may contain teeth. Neurofibromas are seen in the back near the spine, are round and are unilateral. Localized empyema or effusions usually follow pneumonia. Aneurysms show connection with aorta, pulsation, and may have positive blood test.

Primary malignant tumors—lymphosarcoma, Hodgkin's disease, thymic carcinoma. The lymphosarcomas arise from lymph nodes, are multiple, radiosensitive, show difficulty in swallowing and displace esophagus. Hodgkin's disease shows multiple or single nodular enlargments, which are usually bilateral, have no pulsations, may infiltrate lungs, may have other glandular enlargements and are radiosensitive. Primary carcinomas arise from bronchi, esophagus or thymus, are unilateral, extend outward from hilum, produce atelectasis, often show fluid.

Tuberculous mediastinitis is an irregular glandular enlargement, found in any mediastinum, usually shows tuberculosis elsewhere in lungs or glands, or spine, and may have accessible glands for biopsy in the neck.

Lymphatic tumors tend to disappear with irradiation. No radiosensitivity in benign tumors and aneurysm. Slight, if any, sensitivity in carcinoma. Be careful with radiotherapy if tuberculosis is suspected.

TUBERCULOUS TRACHEOBRONCHITIS

RADIATION THERAPY*

By LOWREY F. DAVENPORT, M.D.
BOSTON, MASSACHUSETTS

TUBERCULOUS lesions of the tracheobronchial tree have received little attention until the past decade. The pathology of the lesions has been clarified by Eloesser.[5] Clerf[4] in 1931 removed tuberculosis from the list of contraindications to bronchoscopy and advocated diagnostic examination of all tuberculous cases where the clinical symptoms suggested bronchial obstruction. More recent papers dealing with therapy have suggested local treatment of the lesions.

Radiation therapy has been dismissed as of doubtful value, although few authors give first-hand reports. The purpose of this paper is to call attention to a type, or perhaps more accurately a stage of bronchial tuberculosis where radiation therapy is a logical procedure. In a small number of our cases, such therapy has given moderately encouraging results.

INCIDENCE

Jenks[6] has recently published a review of recent papers dealing with this problem. The reported incidence varies widely with the type of study. Bugher, Littig and Culp[2] found lesions in the bronchi in 41 per cent of cases dying from pulmonary tuberculosis. McIndoe and his associates[8] report demonstrable lesions in 11 per cent of 272 patients examined routinely on admission to the sanatorium. In our group of patients there were 105 cases examined because of suggestive evidence of bronchial disease and 25, or 23.8 per cent, showed gross lesions at bronchoscopy.

TYPE OF LESION

There is general agreement as to the usual variety of lesions as found on bronchoscopic examination. The various types or stages of this disease must be kept clearly in mind. Certainly the prognosis, and perhaps the indicated therapy, varies with the type, extent and location of the lesion.

Most frequently found, both at bronchoscopy and at postmortem, are discrete tubercles usually associated with shallow and circumscribed ulcerations. Such lesions are found about bronchial orifices draining active disease. The predominance of these lesions on the posterior bronchial wall suggests a direct implantation.

Healed lesions with puckered edges, frequently causing more or less fibrotic stenosis of the bronchus, are next most frequent. Such lesions are found predominantly in the more chronic fibroid tuberculosis, where the parenchymal lesion is stationary or inactive. This fibrotic stenosis probably represents an end stage of the first group.

The most serious and fortunately less frequent lesions are those with widespread tuberculous infiltration of the mucous membrane. There is edematous congestion, exuberant granulation, and often extensive fungating ulceration. These cases present the real problems of tuberculous tracheobronchitis. In our experience such fulminating involvement of the bronchial mucosa has been found exclusively in women and preponderantly in young women. Parenchymal tuberculosis in females in the younger age groups commonly runs a fulminating course. We believe that a rapidly spreading bronchial tuberculosis represents, similarly, a lowered general resistance rather than a particular type of lesion.

ROENTGEN PATHOLOGY

Peirce and Curtzwiler[9] have recently given an excellent summary of the roentgenological findings in bronchial tuberculo-

* From the Middlesex County Sanatorium and Pulmonary Clinic of the Massachusetts General Hospital. Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

sis. We agree that atelectasis is the cardinal sign of bronchial obstruction. Other helpful observations are: collapse out of proportion to the amount of pneumothorax; obstructive emphysema, especially in cavities; and lack of correlation between severe clinical symptoms and minor roentgenological changes. Demonstrable bronchial abnormalities on plain films have not been observed in our series.

COURSE AND PROGNOSIS

Bugher, Littig and Culp[2] report that 47 per cent of patients having ulcerative or stenotic lesions died within one year after the diagnosis was made. On the other side of the picture, Warren, Hammond and Tuttle[14] report a group of 57 patients with ulcerative lesions treated with cautery some time during a two year period. There were but 2 deaths in this series and 59 per cent of the cases were healed. There are similarly wide discrepancies in prognosis as reported by others.[6]

At the Middlesex County Sanatorium more than half the cases of pulmonary tuberculosis, examined postmortem, show bronchial lesions. These are usually secondary to extensive parenchymal disease and may be likened to laryngeal involvement. The prognosis in such cases depends almost wholly on the lung involvement and bronchial lesions may be considered an incidental finding.

Among living patients small ulcerations near the bronchial orifice draining active disease are encountered frequently. Where such incidental lesions were discovered and no obstruction to the airway found, local treatment was not instituted.

Bronchoscopy, because of suggestive symptoms of obstruction, disclosed 8 such cases. Discrete ulcers with punctate tubercles were seen but no organic obstruction to a main bronchus encountered. The wheeze was ascribed to viscid secretions. Treatment was instituted for the lung disease without reference to the bronchial lesion. None of these cases has developed symptoms of bronchial obstruction.

A fibrotic stenosis of varying degree was found in 6 cases, excluding those where the distortion was thought to be due to a previous thoracoplasty. Three have symptoms which can be ascribed wholly to the lung pathology. Three show intermittent bouts of fever with each superimposed respiratory infection indicating bronchial occlusion when the already narrowed airway becomes edematous.

In a group to themselves are 11 females whose chief tuberculous lesion was bronchial, and in whom the chief symptoms were due to obstruction. Bronchoscopically, a fearsome picture is presented of engorged mucous membrane, fungating ulcerations, exuberant granulation tissue and caseous exudate. As the course and prognosis vary with the degree of obstruction these cases are reported in detail.

TREATMENT

Samson, *et al.*[10] reported in 1937 a series of cases treated with irradiation. In a later report Samson[11] states that 5 cases with chronic hyperplastic and granulo-ulcerative "lesions" showed retrogression following irradiation. Warren, Hammond and Tuttle[14] report 57 cases treated by cautery and state that 34 were healed, presumably as a result of treatment. Shipman[13] has reported surprising results with ultraviolet therapy but the relationship between the therapy and the results, as he admits, is not clear.

We do not feel that the discrete ulcerations need treatment. We have abundant postmortem evidence and some clinical data to indicate that such incidental lesions heal spontaneously if the parenchymal disease is retrogressive.

The fibrostenotic cases present a recurrent problem. We have carried out dilatation and aspiration in such cases when the symptoms warranted. Benedict[1] has reported excellent palliative but temporary results in such cases. Jones and Dolley[7] have recommended lobectomy and pneumonectomy in selected cases. Churchill[3] has successfully performed a total pneumonec-

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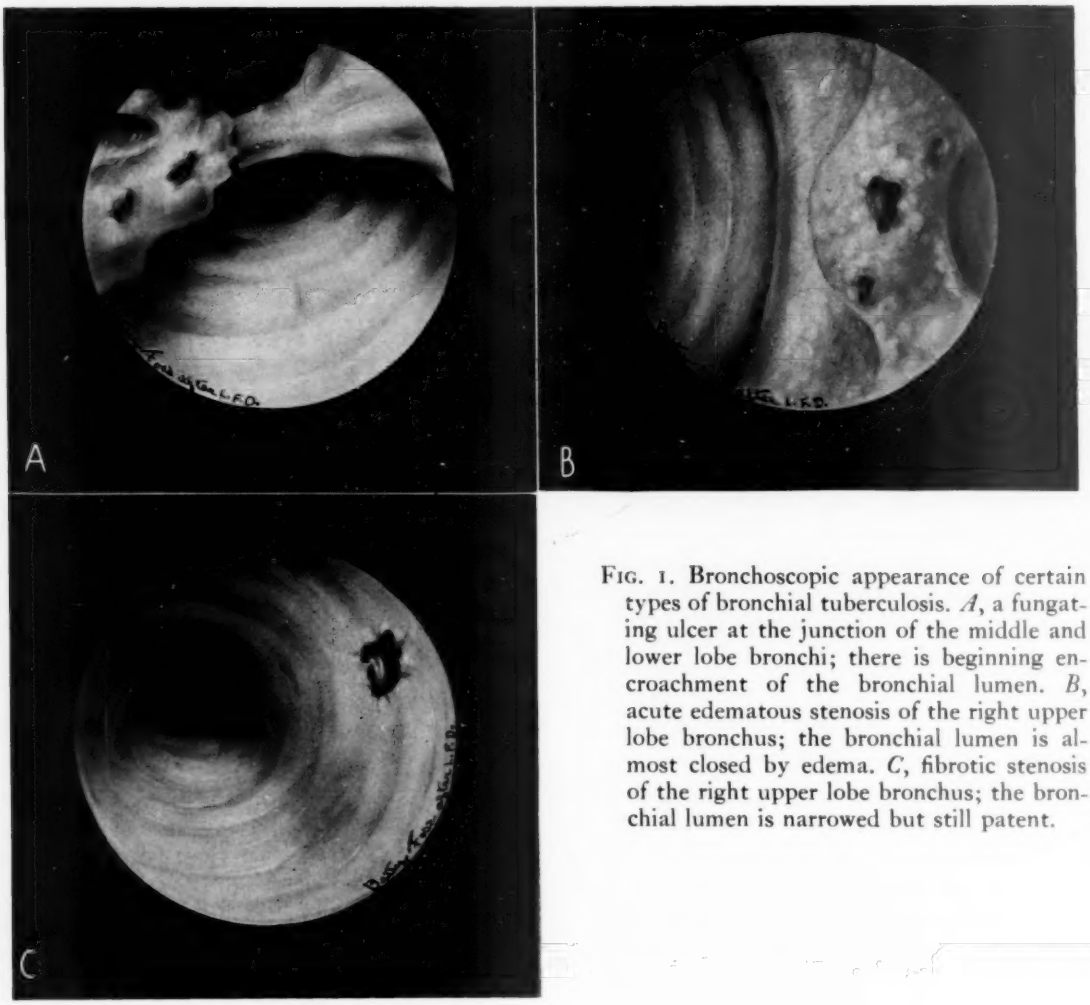

Fig. 1. Bronchoscopic appearance of certain types of bronchial tuberculosis. *A*, a fungating ulcer at the junction of the middle and lower lobe bronchi; there is beginning encroachment of the bronchial lumen. *B*, acute edematous stenosis of the right upper lobe bronchus; the bronchial lumen is almost closed by edema. *C*, fibrotic stenosis of the right upper lobe bronchus; the bronchial lumen is narrowed but still patent.

tomy in one such case observed by us.

The acute fulminating cases present the mechanical problem of obstruction which proves fatal if not relieved. Reasoning from the effects of irradiation on swollen tuberculous glands and fortified by the published reports[10] from Michigan, we were led to try this means of attempting to alleviate obstruction.

DOSAGE

The irradiation was given in courses of six to eight weekly treatments, alternating front and back. The physical factors were 200 kv., 50 cm. skin-target distance; 18 ma., with a total of 840–1,120 r filtered through 2.0 mm. of copper. The portal chosen was centered over the location of the involved bronchus for an area of 15 cm.

RESULTS

Eleven cases of fulminating bronchial tuberculosis with partial obstruction have been discovered in our patients. All were females. There were 6 cases in the third decade, 2 in the fourth and 3 in the early forties.

Seven cases were treated by irradiation in the dosage outlined.

Case I died with increasing stenosis of the involved bronchus and progression of the disease in the contralateral lung.

Case II failed to show bronchoscopic retrogression during treatment but there was never complete obstruction. Subsequently, the lesion has retrogressed and there are now no signs of obstruction.

Cases III, IV, and V showed complete retrogression of the bronchial disease but there has been a flare-up in the parenchymal process. All are young women and the ultimate prognosis is guarded.

Cases VI and VII have shown considerable improvement. There have been no symptoms of occlusion but the ultimate result is in doubt.

Cases VIII and IX were treated with silver nitrate but the treatments were abandoned. Case VIII showed increasing stenosis following cauterization and treatment was abandoned. Case IX had an acute pneumonic spread early in the treatment and the procedure was abandoned.

Case X was untreated as the spread of the disease was so rapid that all hope was abandoned early. Today we would give her a trial with irradiation, but as the postmortem specimen closely resembled treated Case I, it is doubtful as to what we might have accomplished.

An additional case is not reported in detail as treatment is being given elsewhere.

COMMENT

The bronchial obstruction which accompanies certain stages of tuberculous tracheobronchitis is the cause of the grave train of symptoms and the discouraging prognosis in this disease. Shallow ulcerations and discrete tubercles are, in themselves, no serious factor. Treatment to be effective must relieve the obstruction. Bronchoscopic aspiration gives but transient relief. Ultraviolet therapy, empirically recommended for building up the general resistance, may thus be helpful but can have no conceivable local effect on deep tissues.

Cauterization, long abandoned in treatment of tuberculosis elsewhere, may be of some benefit in controlling exuberant granulations. It does not cure tuberculosis anywhere else in the body and the healing of ulcerations following its use may be fortuitous. The report of a case[12] healed in one month following a single application of silver nitrate does not fall into the group of cases which to us have presented the appalling complication. Furthermore, there is considerable danger in repeated bronchoscopic treatment of these acute lesions. Samson[11] reports 3 cases that required tracheotomy following bronchoscopy, all of whom subsequently died. The flare-up reported in Case IX may have been incidental to bronchoscopy, but it is our opinion that the trauma caused by a solid brass tube outweighs whatever advantage cauterization might afford. In addition those cases which really need active intervention show such extensive involvement that topical application through the bronchoscope could not hope to reach the major portion of the disease.

We do not expect any therapeutic triumphs following irradiation. The patient's general resistance will, as always, be the ultimate factor in the outcome. We are accustomed to this viewpoint in collapse therapy and do not expect good results in any patient with rapidly advancing disease. Discouraging results in the treatment of tuberculous tracheobronchitis are to be expected. The mere presence of such a fulminating process is in itself an indication of lowered resistance. If then we can reduce the chances of complete obstruction during the height of this infection, we have accomplished much. In some we shall fail and irreparable lung damage results. Others will be held in check only to succumb later to advancing parenchymal disease. We cannot yet predict how many can be tided over to go on to complete arrest.

REPORT OF CASES

Case I. Z. G., female, aged forty, admitted January 14, 1937, with minimal tuberculosis and sputum heavily positive. There was a wheeze over the right chest with croupy cough. Bronchoscopy March 10, 1937, showed extensive fungating ulceration and edema involving the right main stem bronchus, particularly about the right upper lobe. Right pneumothorax was begun March 20, 1937, and was followed by marked increase in symptoms with increasing wheeze and signs of retained secretions. Bronchoscopy in May showed progression. In July and August she was markedly dyspneic and cyanotic, and had a constant croupy cough. Emergency bronchoscopic drainage was done on several occasions with but temporary relief.

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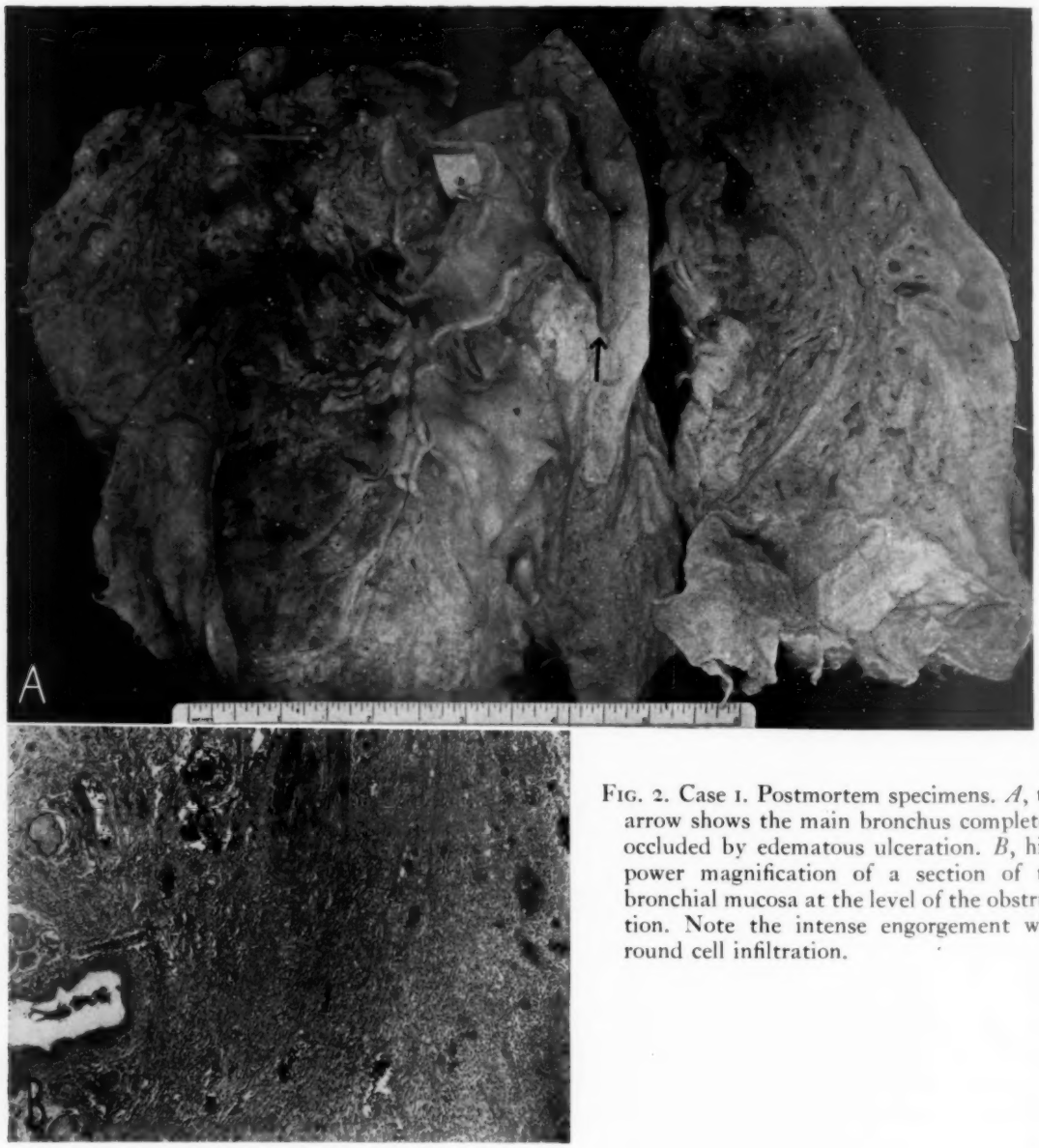

Fig. 2. Case 1. Postmortem specimens. *A*, the arrow shows the main bronchus completely occluded by edematous ulceration. *B*, high power magnification of a section of the bronchial mucosa at the level of the obstruction. Note the intense engorgement with round cell infiltration.

In September, 1937, a course of roentgen treatment was begun. Bronchoscopy in December, 1937, showed disappearance of the ulceration but there was intense engorgement of the mucous membrane with almost complete occlusion of the main stem bronchus. A second course of roentgen treatment was given from December, 1937, to January, 1938. This was followed by decrease in cough. Large quantities of sputum were expectorated indicating better drainage. Her general condition failed to improve. An exacerbation of symptoms developed. The obstruction became complete. The airway was lost.

Postmortem examination showed fibrotic stenosis of the right main bronchus. Bronchiectatic cavities filled with tuberculous pus were found throughout the entire lung. There was extensive tuberculous disease through the entire left lung.

Case II. E. S., female, aged twenty-five, admitted July 7, 1936. A roentgenogram in June showed minimal tuberculosis involving the right upper lobe. Within a month there was complete atelectasis of the right upper lobe which had begun to expand on a routine film

taken six weeks later. Sputum positive. Right pneumothorax was begun August 7, 1936. In spite of an apparently satisfactory collapse, which was partially selective because of atelectasis of the right upper lobe, the sputum remained positive and the patient had a persistent cough and wheeze. Bronchoscopy in May, 1937, showed almost complete occlusion of the right main stem bronchus by edematous mucous membrane and granulation tissue. Ultraviolet therapy was given. Her general condition grew worse. A roentgenogram showed atelectasis of the entire right lung.

Bronchoscopy in November, 1937, showed progression which now involved the trachea. Because of cough and continued secretions, bronchoscopic aspiration was again done in December. Two courses of roentgen treatment were given between December, 1937, and March, 1938. Bronchoscopy, in June, 1938,

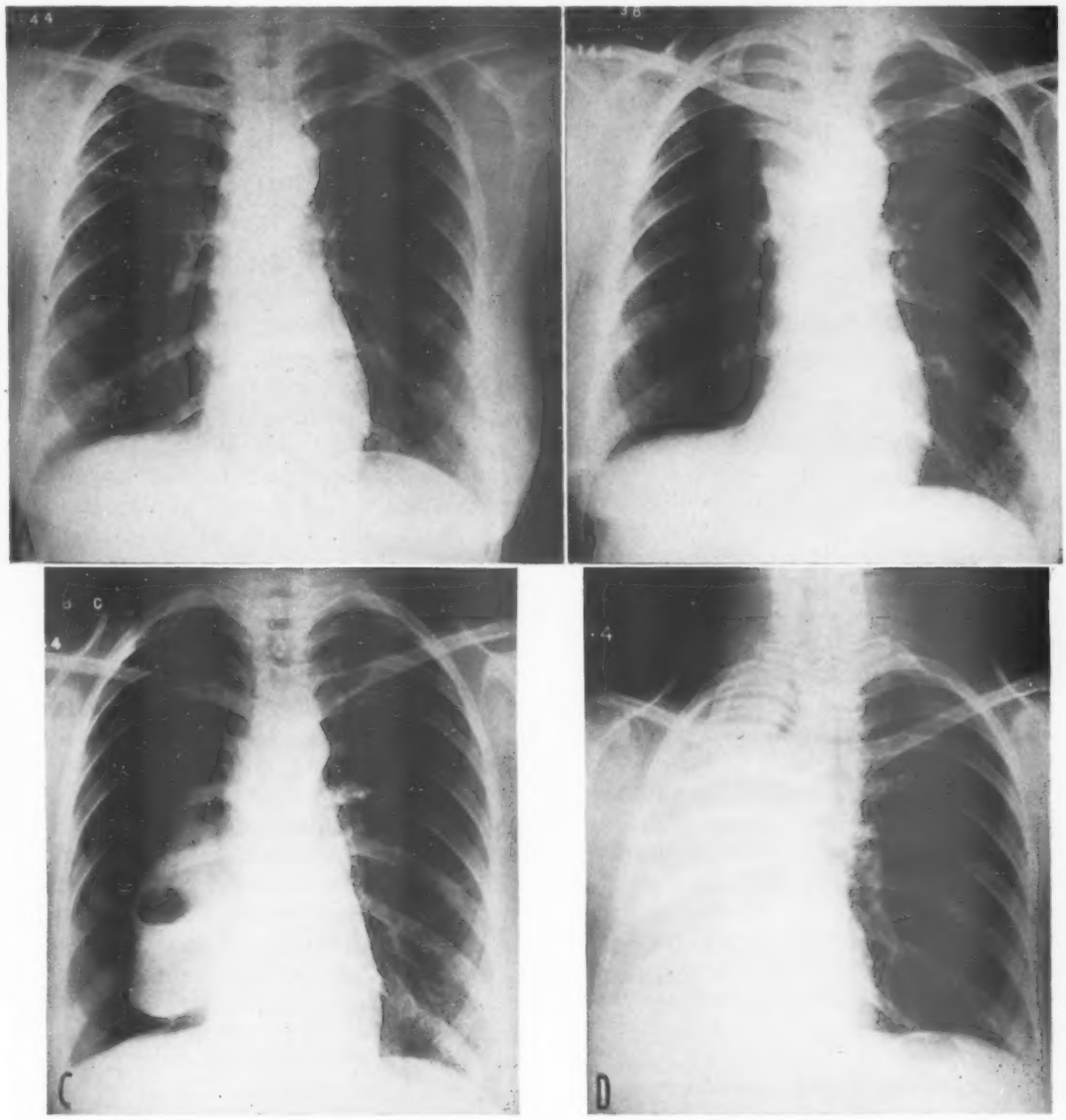

Fig. 3. Case 11. Progressive stages in tracheobronchial tuberculosis. *A*, June 8, 1936. Minimal tuberculosis in the right upper lobe. Unilateral wheeze on physical examination. *B*, July 10, 1936. Complete atelectasis of the right upper lobe. *C*, February 1, 1938. Multiple thin wall cavities developing behind intermittent obstruction. *D*, September 11, 1940. Fibrotic retraction of the right lung. The bronchus has remained open and on direct examination is now free from disease.

showed fungating lesions extending up into the trachea. The right main bronchus appeared completely occluded. Her condition grew worse with persistent fever about 100° F., and a constant croupy cough. A third series of roentgen therapy was given in June and July, 1938. There was little change in her general condition but the right bronchus remained open. The bronchoscope could not now be introduced at the level of the carina. Right pneumothorax was abandoned. The right main bronchus remained open, however, and there was gradual clinical improvement with marked diminution in cough, although the sputum remained intermittently positive for several months. She was discharged in April, 1939. Sputum became negative and follow-up in the clinic has shown a gradual clinical improvement with a gain in weight. The entire right lung field is now obscured by a thickened pleura. The airway has been maintained.

Case III. A. O., female, aged twenty-five, admitted January 25, 1937, with moderately advanced tuberculosis. Sputum negative. Left pneumothorax was begun January, 1937, following which the sputum became positive. A wheeze developed and a roentgenogram showed partial atelectasis of the compressed right upper lobe. Bronchoscopic examination in June, 1937, showed exuberant granulation tissue in the trachea and fungating ulceration in the right main bronchus with marked edema and reddening. The pneumothorax was then abandoned. A course of roentgen therapy was begun in June and July, 1937. In September, bronchoscopic examination showed considerable clearing of the granulation tissue with an increase in the size of the bronchial lumen due to decreasing congestion. Two subsequent courses of roentgen therapy were given between September, 1937, and January, 1938. Bronchoscopy April, 1938, showed marked improvement. There were but a few punctate tubercles in the right main bronchus. Mucosa was pale, not otherwise abnormal. There was no stenosis. Her general condition gradually improved and she was discharged in December, 1938, sputum negative. Serial roentgenograms had shown lobular atelectasis during the course of pneumothorax. Following abandonment of pneumothorax in 1937 there was gradual re-expansion of the right lung. Further roentgenograms showed progression of the parenchymal disease on the right and she was re-admitted to the sanatorium March 8, 1940. Bronchoscopy March 23, 1940, showed an entirely normal bronchus on the right. All the present symptoms were ascribed to parenchymal disease and right pneumothorax was begun March 25. Bronchoscopy July 20, 1940, again showed a normal right bronchus. Pneumothorax is being continued. The sputum is intermittently positive. General condition satisfactory. The airway has been maintained.

Case IV. W. F., female, aged seventeen, admitted May 25, 1937, with a minimal tuberculosis. Sputum negative. Right pneumothorax begun June, 1937, followed by wheeze and intermittent fever suggesting bronchial obstruction. Sputum became positive and cough was continuous and of a croupy nature. The pneumothorax was abandoned as each refill was followed by symptoms of obstruction. Bronchoscopy January 15, 1938, showed exuberant waxy granulation tissue in the right main bronchus, particularly about the upper lobe opening. A course of roentgen treatment was carried out in February and March. Bronchoscopy April 20, 1938, showed complete clearing of the caseous granulation tissue. Her general condition improved for a time but serial roentgenograms showed progression of the parenchymal disease and the wheeze again developed.

Bronchoscopy September 7, 1938, showed marked injection of the right main bronchus with edema and small punctate ulcerations surrounded by exuberant granulation tissue. A second course of roentgen treatment was begun. Bronchoscopy in January, 1939, showed no edema nor ulceration tissue but there was some hypertrophic mucous membrane about the middle lobe opening. Serial roentgenograms showed an increase in the parenchymal involvement coincident with the appearance of the wheeze. Subsequent examinations showed progression in the spring of 1938 with gradual retrogression subsequently. There had been intermittent lobular atelectasis in the region of the right middle lobe. The cough has now completely subsided. There is no further wheeze. Sputum is negative but a recent roentgenogram has shown progression of the parenchymal disease; though her present condition is considered poor, the airway has been maintained.

Case V. H. P., female, aged twenty-nine, first admitted March 15, 1938, with minimal tuberculosis. Sputum negative. Left pneumo-

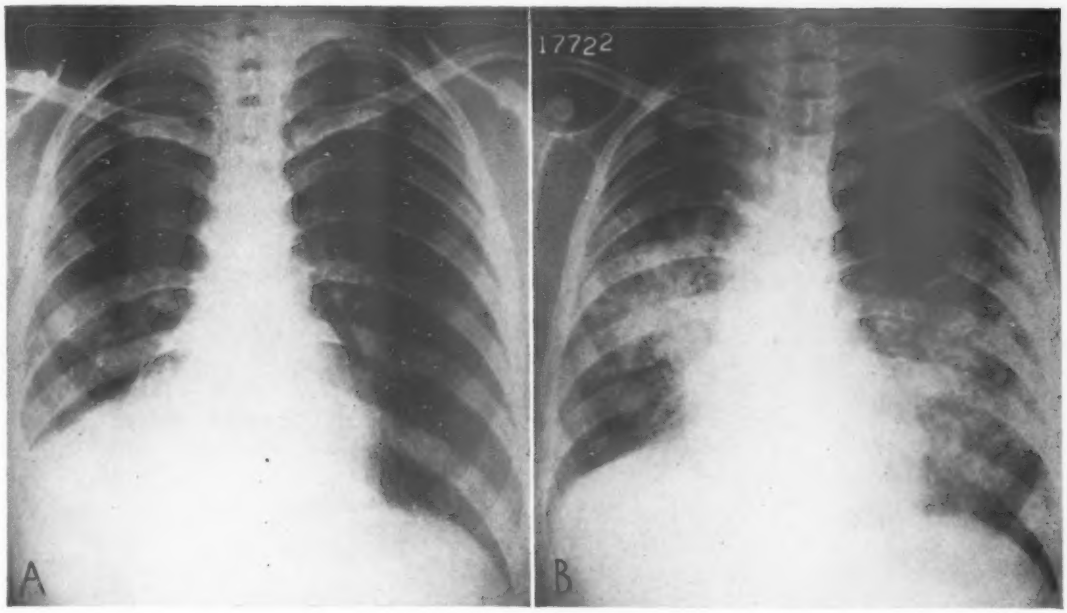

Fig. 4. Case x. Fulminating parenchymal disease associated with tracheobronchial tuberculosis. *A*, October 5, 1939. Collapse of the right lower lobe. Direct examination showed a fungating ulceration in the bronchus leading to this lobe. *B*, October 16, 1939. Extensive bilateral progression eleven days later.

thorax begun March 28, 1938. Left pneumolysis July 13, 1938. She was discharged and remained asymptomatic until 1939 when she developed a heavily positive sputum and a wheeze. Roentgenograms showed a clear right lung and there was an apparently satisfactory pneumothorax on the left. Bronchoscopy May 31, 1939, showed involvement of the upper third of the left main bronchus with exuberant granulation tissue and grayish-white caseous exudate. The mucous membrane looked as though it had been charred with caustic but there was no appreciable obstruction to the airway. The pneumothorax was continued and because of increasing cough and wheeze she was again bronchoscoped October 7, 1939. There had been marked progression. There was now exuberant granulation tissue through the lower third of the trachea with marked engorgement of the mucous membranes and thickening of the carina. The edematous granulations about the left main bronchus almost occluded this opening. The left pneumothorax was discontinued and she was given a course of ultraviolet therapy. The symptoms of obstruction increased.

The next bronchoscopy January 27, 1940, showed complete occlusion of the left main bronchus with pearly-white tuberculous tissue which appeared almost like gristle and an increase in the process involving the trachea. A course of roentgen treatment was given and bronchoscopy April 27 showed but a slight change. A second course of roentgen therapy was begun in May and was followed by a definite improvement in the clinical symptoms.

The last bronchoscopy September 14, 1940, shows surprising improvement over the previous examination. All the caseous exudate had disappeared. The mucous membrane was still injected and edematous but air entered freely into the lung beyond. Serial roentgenograms have shown the re-expanding left pneumothorax with mottled infiltration through the left lung associated with a diffuse pleurisy. The airway has been maintained.

Case vi. E. K., female, aged thirty-two, readmitted April 12, 1940, with a ten year history, developing insidiously and culminating in hemoptysis in 1933. She was examined March 29, 1934. There was moderately advanced bilateral disease. Sputum positive. Left pneumothorax begun May 10, 1934. Discharged May, 1935. Sputum negative. Pneumothorax refills were discontinued in July, 1939. She was considered an arrested case until the spring of 1940. Examination in the clinic March, 1940, disclosed a wheeze and a positive sputum. Bron-

choscopy April 27, 1940, showed marked displacement of the trachea to the left with swelling and a distortion of the carina. The mucous membrane was edematous and showed exuberant granulation tissue. The left main bronchus was almost completely occluded but admitted a small aspirating tube and purulent secretions were obtained from below the point of obstruction. Eight roentgen treatments were given in May and June. Bronchoscopy August 3, 1940,

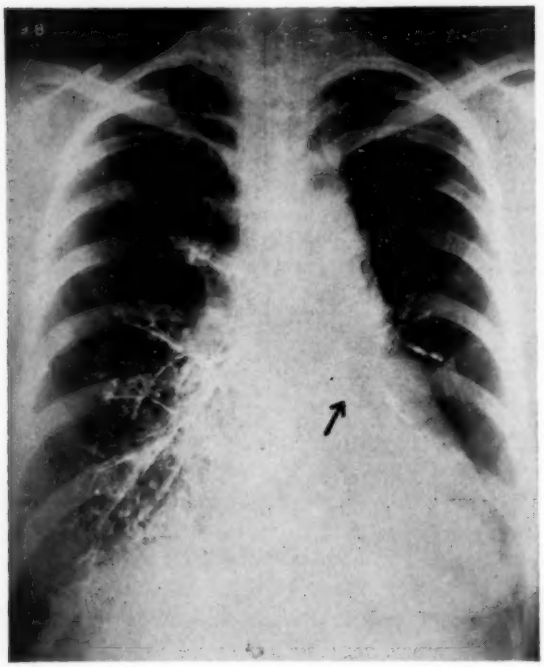

Fig. 5. Iodized oil instillation in a case of fibrotic stenosis. The arrow shows the level of the obstruction. The lower lobe is airless but the obstruction is complete only with superimposed acute infection.

showed marked improvement. There was no edema or injection. Mucous membrane was pale and showed no ulceration. The lumen itself was about one-third normal size. The stenosis appeared to be predominantly fibrotic. Sputum is still positive. Roentgenograms show complete clouding of the left lung with atelectasis and associated pleuritis. General condition improved. Wheezing has disappeared. The airway has been maintained.

Case vii. L. B., female, aged thirty-nine, admitted October 30, 1939. Insidious onset in 1937 when roentgenograms showed a minimal fibrosing lesion. Acute pleurisy with high fever developed in September, 1939. On admission the sputum was positive. The roentgenogram showed a moderately advanced tuberculosis involving predominantly the left lung. Left pneumothorax begun December 12, 1939. The collapse was satisfactory but the sputum remained positive and a persistent wheeze developed. Bronchoscopy May 18, 1940, showed intense edema and injection of the left main stem bronchus which was reduced to 50 per cent its normal size. There was grayish-white caseous exudate over the bronchus for a distance of several centimeters. There was no fungating ulceration. Eight roentgen treatments were given in June and July. At the time of her last examination she had gained 10 pounds in weight. Sputum was positive but not heavily so. Wheezing disappeared. The airway has been maintained.

Case viii. L. H., female, aged forty-four, admitted October 9, 1934. The roentgenogram showed minimal tuberculosis. Sputum positive. There was a wheeze over the right chest. Right pneumothorax begun in November, 1934. Internal pneumolysis in July, 1935. In spite of a satisfactory collapse the sputum remained positive and the wheeze became more pronounced. Bronchoscopy at an outside hospital showed extensive bronchial tuberculosis, in May, 1936. Three treatments with silver nitrate were given at the Peter Bent Brigham Hospital in 1936. Sputum remained positive and there was increasing stenosis of the right main bronchus with atelectasis of the right lung. Course was variable and accompanied by bouts of fever due to retained secretions. Bronchoscopy here in March, 1937, showed fibrotic stenosis of the right main bronchus associated with exuberant granulation tissue causing almost complete occlusion. Subsequent bronchoscopies have shown no change in the degree of stenosis. Sputum remains intermittently positive. Her symptoms are variable depending on the amount of retained secretions. Serial roentgenograms now show a completely atelectatic right lung with the underlying parenchyma obscured by thickened pleura. The airway was temporarily lost with irreparable damage to the lung.

Case ix. H. M., female, aged twenty, admitted June 26, 1940. The roentgenogram showed moderately advanced tuberculosis involving the right lung. Sputum positive. Physical examination showed a unilateral wheeze. Bronchoscopy July 6 showed partial occlusion

Fig. 6. Postmortem specimen in advanced tuberculosis showing the discrete ulcer without edema about the upper lobe orifice. This is a frequent finding in advanced disease and gives rise to no symptoms.

of the right upper lobe bronchus with extensive tuberculous granulation tissue and caseous exudate. On July 13 and 20 the localized lesion about the right upper lobe was cauterized with 30 per cent silver nitrate. About one week after the second application the patient developed a chill and fever to 102° F. The roentgenogram showed a dense pneumonic reaction through the right upper lobe without atelectasis. It was thought that her acute symptoms were due to the development of a tuberculous pneumonia. There was obviously not complete stenosis so further treatment was omitted. Her general condition is unsatisfactory and the parenchymal process is spreading.

Case x. P. T., female, aged twenty, admitted January 29, 1939. Acute onset previous July diagnosed as pneumonia. Roentgenograms at the Massachusetts General Hospital in December, 1938, showed partial atelectasis of the right lower lobe. Bronchoscopy was reported as showing acute edematous stenosis. On admission the sputum was positive. The roentgenogram showed involvement of the right middle and lower lobes with new productive disease on the left. Bronchoscopy February 3, 1940, showed stenosis of the right main stem bronchus without ulceration. There was edema and congestion at this level. Patient ran a progressive course from the time of admission with increasing stenosis as evidenced by serial roentgenograms and extensive rapidly spreading infiltration on the left. The right airway was lost and the obstruction became complete. Death occurred March 13, 1940.

Postmortem examination showed almost complete stenosis of the right main bronchus below the upper lobe opening. Beyond this point the lung was airless with extensive bronchiectasis and multiple small cavities containing purulent secretions.

SUMMARY

Ten cases of fulminating tuberculous tracheobronchitis are reported. The grave symptoms in all, and death in two, can be attributed to obstruction, complete or partial, of the bronchial airway with retained secretions. Radiation therapy was tried in seven of these ten patients. One died with clinical symptoms and postmortem evidence of complete obstruction. Six cases have been followed through the acute congestive stage without developing complete obstruction. All are improved. None is completely well.*

* For discussion see page 517.

Acknowledgment: The radiation therapy was given under the direction of Dr. Merrill C. Sosman, Roentgenologist to the Peter Bent Brigham Hospital, whose aid made this study possible. The postmortem data were furnished by Dr. Reuben Schulz, Consulting Pathologist to the Middlesex County Sanatorium.

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SARCOID DISEASE AS REVEALED IN THE CHEST ROENTGENOGRAM*

By DONALD S. KING, M.D.
BOSTON, MASSACHUSETTS

SARCOIDOSIS is a disseminated disease which involves particularly the skin, lymph nodes, lungs, eyes, bones and spleen, though it may affect almost any organ in the body. It is the lupus pernio of the dermatologist, the uveoparotitis of the ophthalmologist, the cold or torpid form of disseminated tuberculosis of the phthisiologist, the osteitis tuberculosa multiplex cystoides of the orthopedist, and a few other syndromes rolled into a single disease which looks like tuberculosis but does not act like it since sarcoid runs a comparatively benign course. Histologically the lesions are like those of tuberculosis even to the presence of giant cells, but there is no caseation, tubercle bacilli are very rarely found either by staining methods or animal inoculation, and tuberculin tests even with strong dilutions are usually negative. Some writers maintain that sarcoid disease is simply non-caseating tuberculosis such as occurs after the injection of tubercle bacilli into a special host like the rat; and others, that it is caused by a specific virus as is lymphogranuloma inguinale. In this paper I cannot discuss these moot points but will give certain highlights of our clinical study of this interesting syndrome.

The diagnosis files of the Massachusetts General Hospital, the Massachusetts Eye and Ear Infirmary, and the Middlesex County Sanatorium show that since 1921 the diagnosis of sarcoid disease has been seriously considered in 142 cases but established in only 112. Since as roentgenologists you are particularly interested in the chest lesions I have selected from the 112 only those in which the chest roentgenogram shows shadows which we consider characteristic of sarcoid and in which the clinical evidence justifies this diagnosis. There are 50 such cases. In 24 of these the diagnosis is proved by biopsy of skin or node; in 11 there are clinical sarcoid lesions of eyes, skin, glands or bones but no biopsy; and in the other 15 we have only the chest roentgenogram as evidence. I have not included in this series 7 cases diagnosed erythema nodosum or rheumatic fever in which the chest roentgenogram is exactly like that of sarcoid.

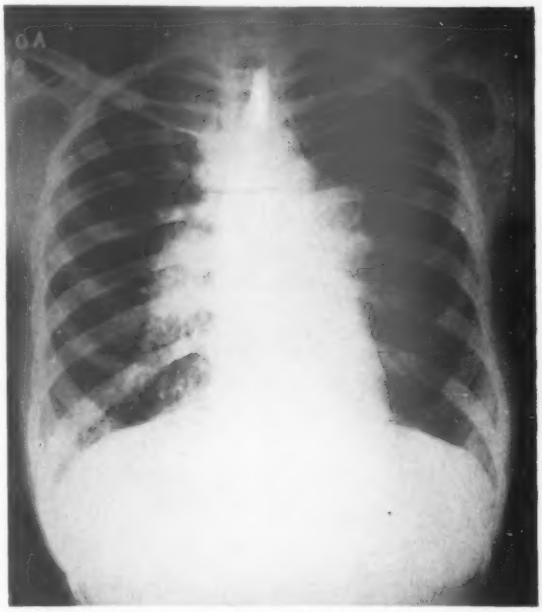

FIG. 1. Enlarged mediastinal nodes in a case with sarcoid lesions of the iris.

In order that we may understand each other let me show quickly what we consider characteristic films. The process may be confined entirely or mainly to the hilar nodes (Fig. 1). It may involve only the lung fields giving an appearance indistinguishable from miliary tuberculosis (Fig. 2). (This film is of the same patient as the previous one but taken a year later.) It may involve the nodes and apparently radiate from them into the lung fields (Fig. 3).

* Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

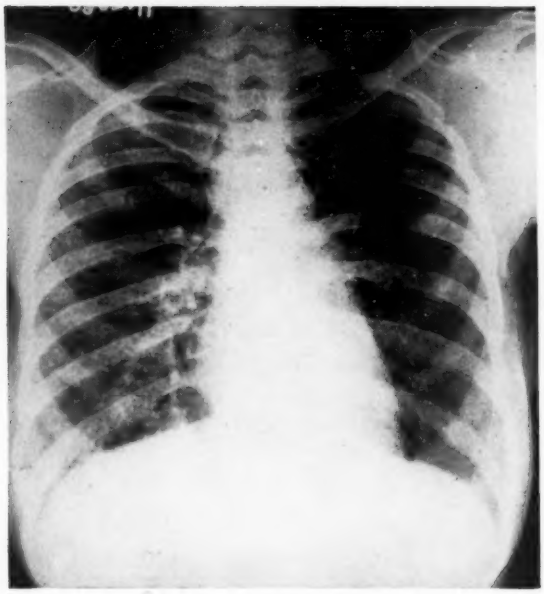

Fig. 2. Miliary sarcoid lesions in the lungs without enlarged nodes. (Same case as in Figure 1 one year later. Three years later chest roentgenogram was normal.)

Then there are more extreme degrees of these forms (Figs. 4 and 5).

As a clinician speaking to roentgenologists I should like to reëmphasize the fact that the chest roentgenogram is not a measure of the gravity of the disease. Extensive pulmonary changes may clear without ever giving respiratory symptoms and be accompanied by only slight evidence of a disseminated process. On the other hand,

Fig. 3. Characteristic enlargement of the mediastinal nodes in sarcoid disease. (Sixteen months later chest roentgenogram showed marked clearing.)

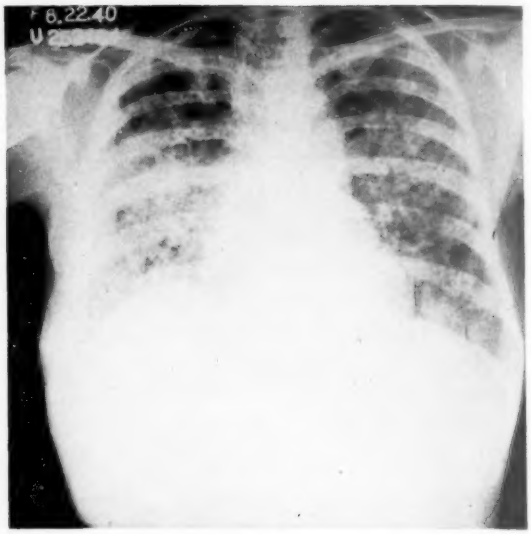

Fig. 4. Unusually marked pulmonary involvement in sarcoidosis proved by biopsy of an axillary node.

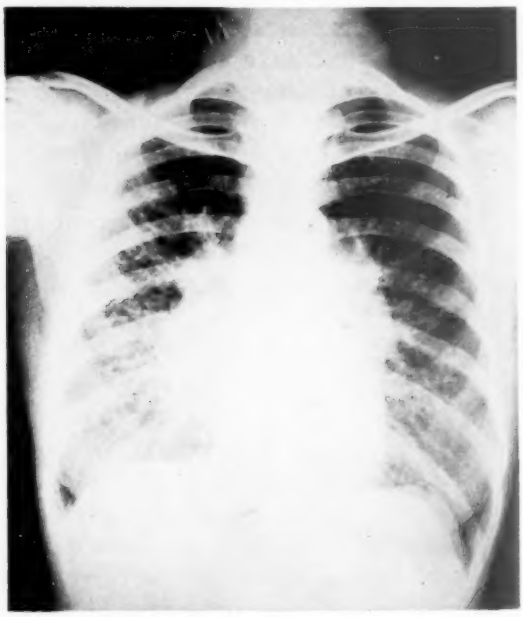

Fig. 5. Sarcoid disease of the hilar nodes with involvement of the surrounding pulmonary tissue.

there may be little chest involvement as shown in the roentgenogram but lesions in other organs which cause prolonged and serious disability. Let me illustrate the protean nature of this disease by roentgenograms from our sarcoid gallery.

This patient (Fig. 6) had roentgen shadows out of all proportion to his mild respiratory symptoms. He was not exposed to silica but because of the roentgen changes

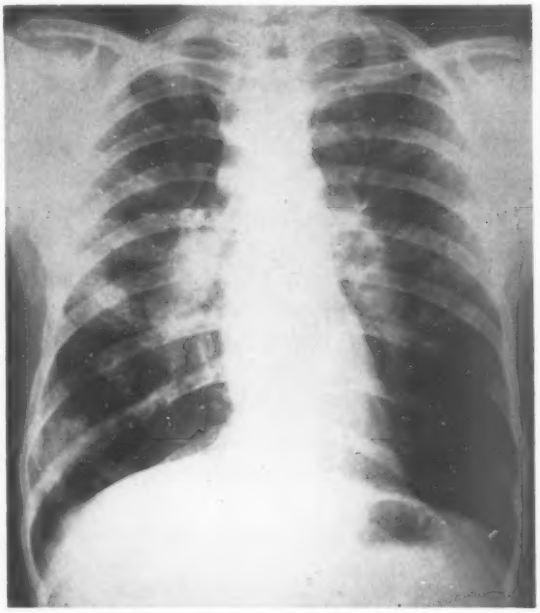

Fig. 6. Unproved sarcoid disease with few pulmonary or other symptoms.

he thought of suing his company for disability due to silicosis. Ten months later, however, a new roentgenogram (Fig. 7) spoiled the evidence because the lesions had completely disappeared.

The next case with extensive bilateral pulmonary lesions (whose film is shown in Fig. 4) did have accompanying severe symptoms of paroxysmal cough, expectoration, night sweats and dyspnea.

In the next case (a colored woman) acute dyspnea and wheezing were the chief complaints. The chest roentgenogram shows enlarged mediastinal nodes which were thought to be narrowing a bronchus and causing the symptoms, but at bronchoscopy the mucous membrane was seen to be

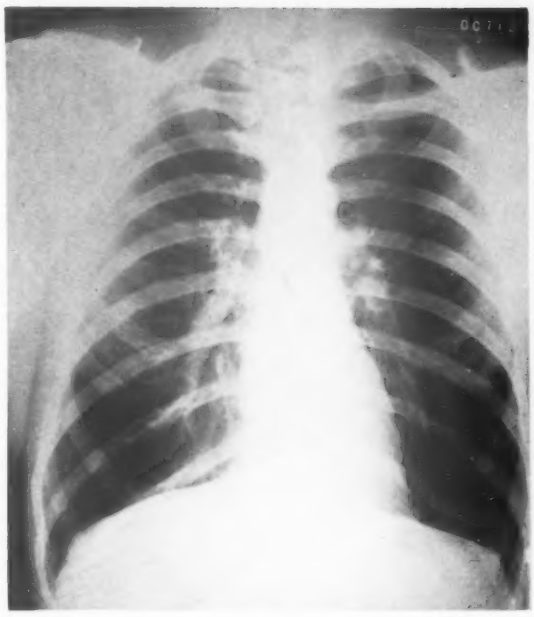

Fig. 7. Same case as Figure 6 ten months later with complete clearing.

infiltrated by lesions proved sarcoid on biopsy. This case had in addition marked enlargement of the cervical lymph nodes. In many instances such a finding is the chief complaint as in the group of colored people reported by Longcope and Pierson.[1]

My next case is one of sarcoid of the skin similar to those described by Besnier[2] in

Fig. 8. Bone cysts in a case of extensive "lupus pernio" or sarcoid of the skin.

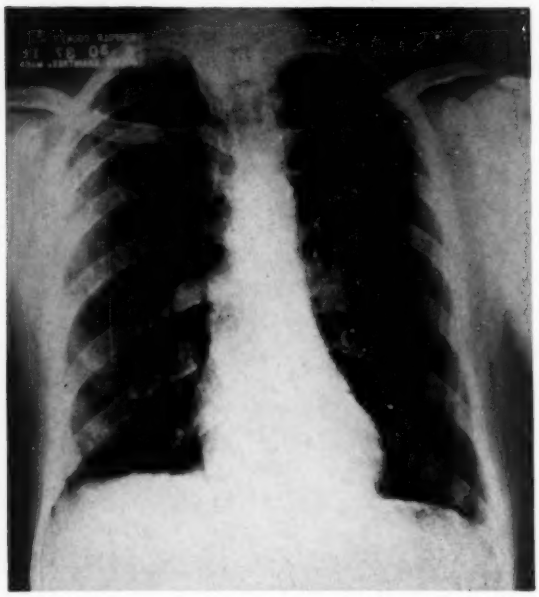

Fig. 9. Pulmonary lesions in a case of proved sarcoid of the spleen and peritoneum. (Three years later lung fields were almost clear.)

1889 under the name of "lupus pernio." Observation of our patient began in 1915 when she was seventeen years old. Her first symptom was painless swelling of the fingers diagnosed at the time as tuberculous dactylitis and requiring amputation of three fingers. In 1920 roentgenograms of the fingers showed no bone lesions, but skin lesions had appeared, and a chest roentgenogram showed enlarged hilar nodes. By 1936 true sarcoid bone cysts were evident (when they first appeared we do not know) (Fig. 8), and a chest film showed only scattered shadows in the lower lung fields. At the present time marked soft tissue swelling of the fingers persists; there are still extensive areas of involvement of the skin of the face, nose, eyelids and right arm; the disease has now attacked the vocal cords causing marked hoarseness; and the chest film shows no change.

Figure 9 shows a diffuse pulmonary process, but the patient who had this process came into the hospital because of a large abdominal mass. Exploratory operation revealed a huge spleen full of miliary lesions and similar lesions on the peritoneal surface. The spleen was removed and diagnosed by the pathologist as sarcoid, not tuberculosis. Now, three years after operation, the patient complains only of asthma from which he has suffered all his life. His recent chest roentgenogram shows the lung fields almost clear.

So much for the illustrative cases.

Although I have made the point that the chest lesions are only part of the whole picture of sarcoidosis, nevertheless they are of importance and we must try to discover what happens to them with the passage of time. Let us return, therefore, to a study of the serial roentgenograms.

Of the 50 cases we have a satisfactory follow-up on 37 (18 from the biopsied group, 10 from the clinical and 9 from the roentgenogram). In 23 of these the lesions seen roentgenographically in the chest have cleared completely or almost completely in from seven weeks to three years. The average time of clearing is twenty-two months. In 3 the process is clearing but has not disappeared entirely after one year, two and a half years, and three years respectively. In

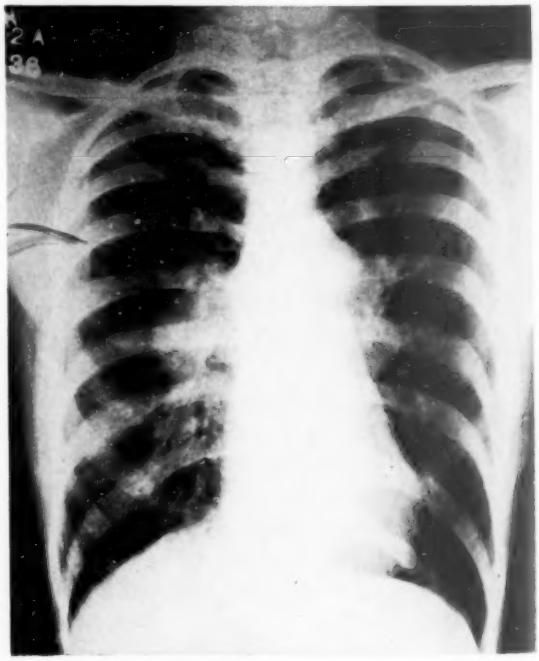

Fig. 10. Hilar nodes in a clinical case of sarcoid. No biopsy.

8 there is no change in from three months to four years. In 3 the process is spreading after seven months, eight months and four years. The last of these "spreads" deserves special mention because after apparent clearing there was a relapse with marked roentgenographic involvement of the lungs but without a corresponding increase in symptoms. The first film taken in 1936 (Fig. 10) shows the characteristic involvement of the hilar nodes. In 1938 (Fig. 11) the lesions had almost cleared, but in 1940(Fig. 12) the process has spread through the lung fields. This is our only case of relapse after apparent clearing. I believe that the process will again clear, but it is possible that this is now active pulmonary tuberculosis.

From the cases reported in the literature one concludes that sarcoid lesions may either resolve or fibrose and that the end stage of a pulmonary process may be a fibrosis of sufficient degree to cause right-sided heart failure and death. In none of our cases is there any evidence that active sarcoid lesions have become fibrotic, and we have only 2 cases of possible pulmonary fibrosis in which there is a suspicion that sarcoid was the underlying etiologic factor. In one case the diagnosis of sarcoid was made from the chest roentgenogram taken in 1934 (Fig. 13). In this film the hilar node enlargement is fairly characteristic. (There were no extrapulmonary lesions for biopsy.) The film taken in 1938 (Fig. 14) shows less prominent nodes with shadows which can be interpreted as fibrosis extending toward the bases. There is no proof, however, that this fibrosis has developed in fresh sarcoid tissue.

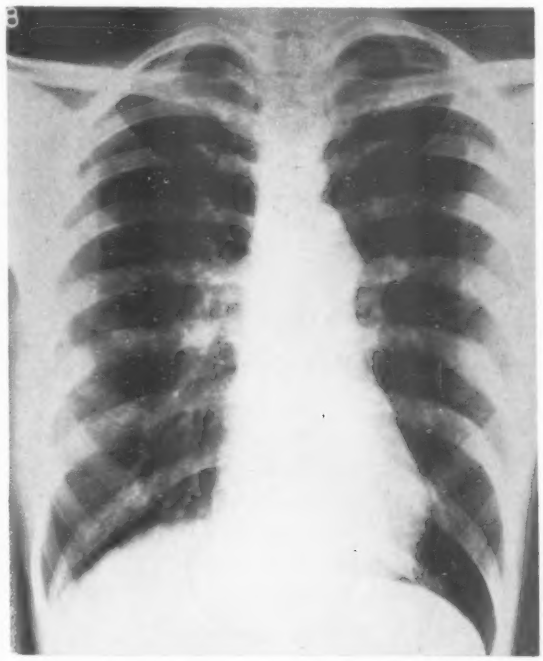

Fig. 11. Same case as Figure 10 two years later showing clearing.

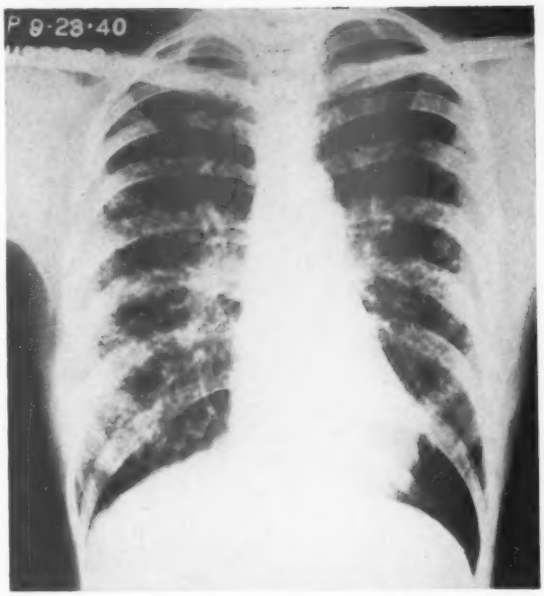

Fig. 12. Same case as Figure 10 four years later with invasion of the lungs.

Our second case which is from the Middlesex County Sanatorium came to autopsy and is therefore of particular interest. In 1919 a 58 ounce spleen was removed and in 1939 a review of the microscopic slides established the diagnosis of sarcoid disease in the spleen. In 1920 the roentgenogram showed a "spot in the lung" which had increased by 1924. At this time there began cough, expectoration and right pleural pain which lasted until death fifteen years later. In 1926 the chest roentgenogram showed "coarse mottling" in the upper third of the left lung and a "small amount" on the right. In 1934 there was "dense infiltration" of the upper half of the left lung with cavity

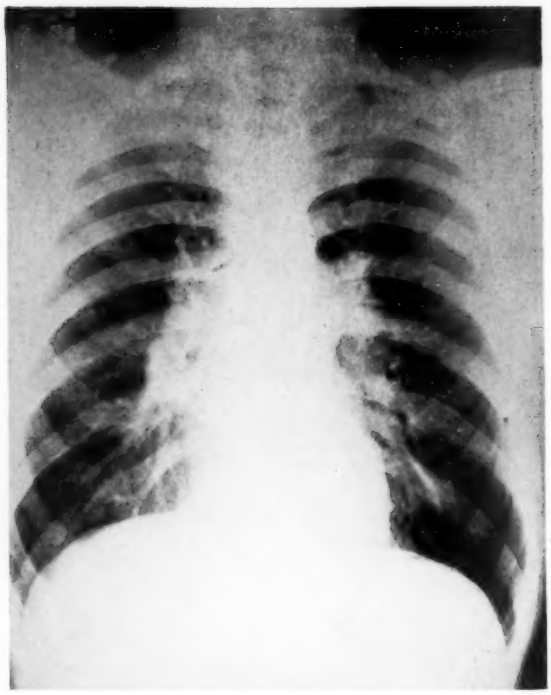

Fig. 13. Unproved sarcoid nodes with suggestive pulmonary fibrosis.

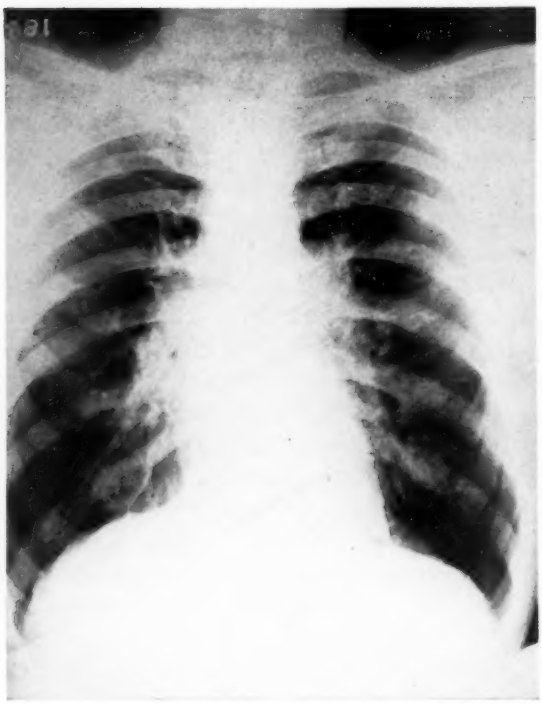

Fig. 14. Same case as Figure 13 four years later with probable fibrosis still present.

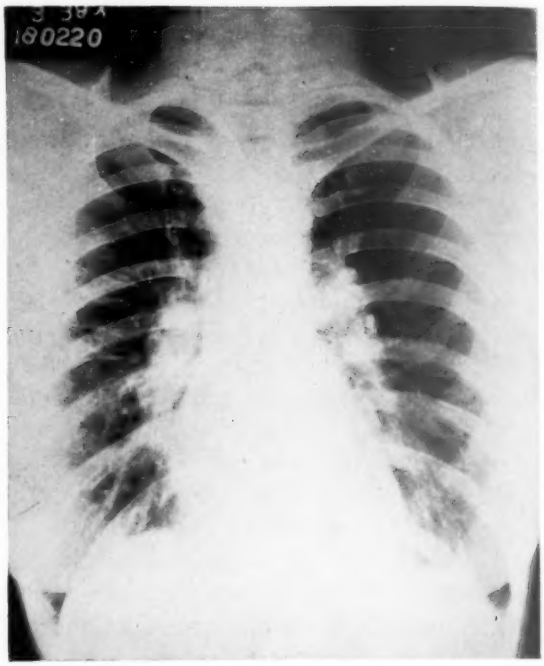

Fig. 15. Proved erythema nodosum with enlargement of hilar nodes like that of sarcoid disease.

formation and much less involvement on the right. In 1938 there was extensive fibrosis throughout the upper two-thirds of both

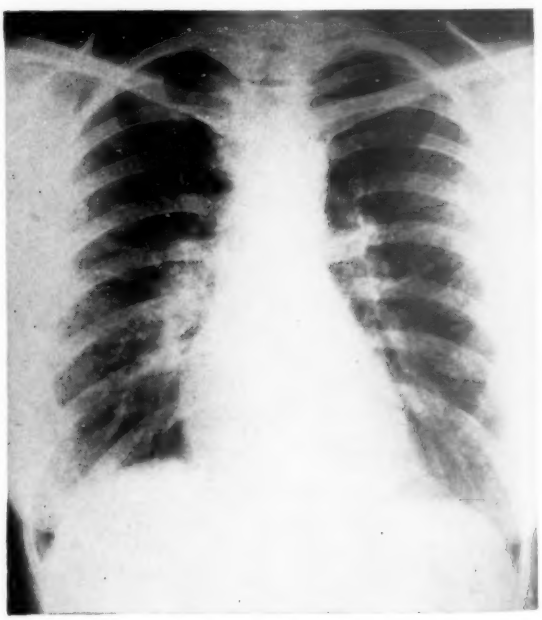

Fig 16. Same case as Figure 15 nine months later with miliary lesions in the lungs.

lungs with large cavities at both apices. The pathologist, Dr. Reuben Schulz, found sarcoid lesions in the lungs, lymph nodes and liver and believes that the sarcoid lesions were responsible for the pulmonary fibrosis either directly or as a result of secondary infection. We believe that the relation of sarcoid to pulmonary fibrosis is still undetermined.

In regard to the prognosis of sarcoid chest lesions, therefore, we conclude that the unmistakable tendency is toward clearing but the time required varies and may be long.

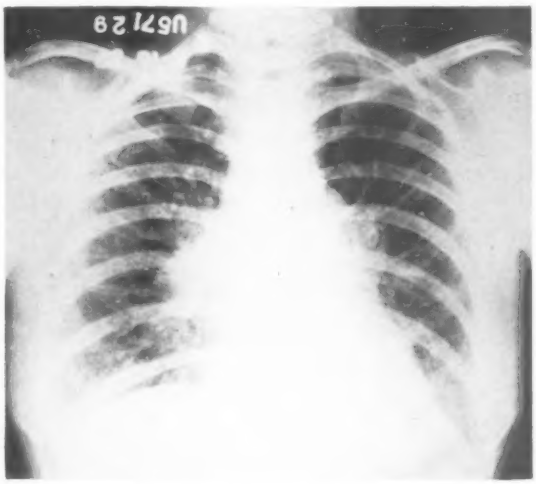

Fig. 17. Proved sarcoid of the skin with chest roentgenogram consistent with sarcoid but sputum full of *Monilia*.

I wish there were time to discuss the differential diagnosis of the chest lesions but can mention only two confusing pictures. The first is erythema nodosum in which the chest roentgenogram may be exactly like that in sarcoid. Figure 15 shows the roentgenogram of a biopsied case with typical hilar lesions. Nine months later (Fig. 16) there was less involvement of the nodes but diffuse lesions in the lung fields. Six months after this both lesions had cleared. The second problem relates to associated mycotic infection. I have reported one case as having cleared in seven weeks and must now confess that I am not sure that the chest lesions were real sarcoid. The biopsy of a

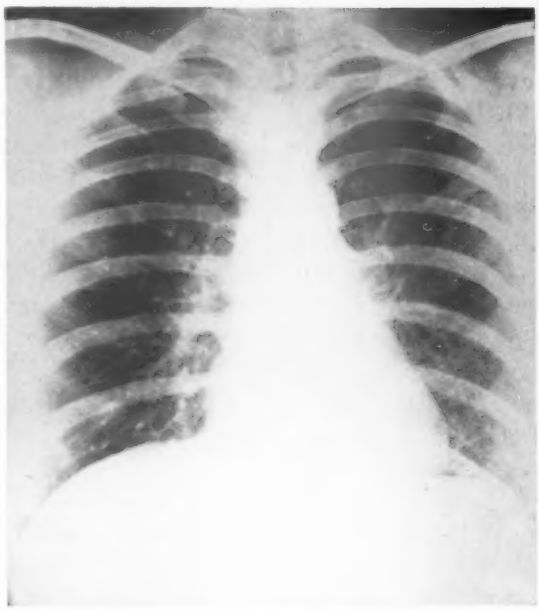

Fig. 18. Same case as Figure 17 seven weeks later after iodide treatment.

skin nodule did show sarcoid disease and the chest roentgenogram (Fig. 17) was considered consistent with that diagnosis, but the sputum was full of *Monilia* and to com-

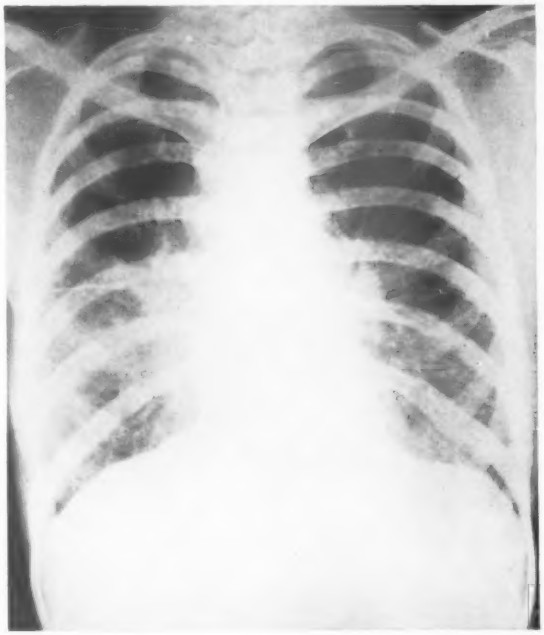

Fig. 19. Proved sarcoid of the skin with *Aspergillus* in the sputum. No change after iodide. Lungs cleared after three years.

plicate matters the basal metabolic rate was +45 per cent. Iodide was given, and in seven weeks the lesions had cleared (Fig. 18). Perhaps they were the lesions of moniliasis. Of course, we hoped we had found a quick cure for sarcoid disease, but iodide "exhibited" in other cases has brought about no noticeable change.

I might mention another case with extensive sarcoid skin lesions and the typical chest roentgenogram (Fig. 19) in which *Aspergillus* was constantly present in the sputum but no benefit derived from iodide. Both skin and chest lesions cleared in three years.

There is one other point I should like to make in connection with our study, though it does not concern the chest roentgenogram. In discussing the diagnosis of sarcoid disease much stress is laid on the importance of routine roentgenograms of the phalanges. Of our 50 cases 35 had roentgenograms of the hands or feet or both. In only 6 were bone lesions found. Five of these were in the group in which sarcoid had been proved by biopsy. In only 1 of the 6 did the roentgenogram show bone lesions which had not been forecast by soft tissue swelling.

In summary, sarcoidosis is a disseminated and protean disease involving in varying degrees the skin, lymph nodes, eyes, bones, lungs and many other tissues. In our experience the lesions in the mediastinal nodes and lungs tend to clear without demonstrable fibrosis, the average time for clearing being twenty-two months, but there are exceptions. Bone lesions were present in a little over 10 per cent of our series. Erythema nodosum gives a chest roentgenogram indistinguishable from that of sarcoid disease, and in two of our cases a possible mycotic infection complicated the picture.*

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* For discussion see page 517.

THE INCIDENCE OF BRONCHOMYCOSIS IN THE SOUTH*

By ROBERT J. REEVES, M.D.
DURHAM, NORTH CAROLINA

THE study of mycology dates back to the year 1677, when Hooke discovered that the yellow spots on the leaves of roses consisted of filamentous fungi. It was not until 1839 when Langenbeck discovered *Oidium albicans*, the fungus of thrush, and found it to be parasitic to man, that the medical profession recognized the existence of fungus disease.

My investigation[9] into the subject of the bronchomycoses has been stimulated by the frequent occurrence of this disease in this particular locality in the South. It is becoming one of the common conditions in the differential diagnosis of vague pulmonary disease. No doubt it is and has been frequently classified as fibroid tuberculosis or listed under the much abused term, chronic intersititial fibrosis. Many cases of chronic mycotic infections are no doubt listed as unknown pulmonary conditions, due to the fact that fungi were not found in the usual routine studies of the sputum.

Smith, Martin and associates[5,6,7,11] of our Department of Bacteriology, have contributed numerous articles describing the technique for cultivation and identification of fungi.

Fawcitt,[3] in 1936, gave a very thorough résumé of the literature and I am indebted to him for his suggestions and help in classifying these various mycoses. He quotes Ramsbottom, the keeper of the botany department of the British Museum, who states that they have on record over one hundred thousand known and named species of fungi. Only the few herein described, however, are pathogenic to man (Fig. 1).

The bacteriological description of the true fungus consists of tubular branched filaments, hyphae. The protoplasm is contained within thin limiting continuous walls. The whole vegetative body of the fungus consists of numerous hyphae, termed mycelium. In addition, a true fungus is

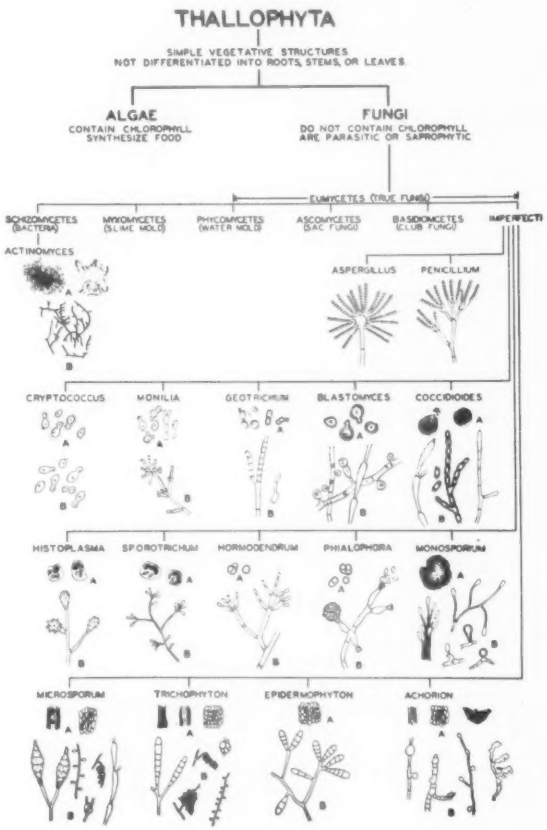

FIG. 1. Showing the various fungi and the general classification of the group imperfecti.

characterized by a special spore-bearing apparatus. The mycelium may be loose and webb-like in structure, as in common molds, or may take the form of a compact mass, produced by the branching and interweaving of the hyphae. In the more highly organized fungi, the hyphae show numerous septa, and the sexual organs and sexual

* From the Department of Radiology, Duke University College of Medicine, Durham, North Carolina. Read at the Forty-first Annual Meeting, American Roentgen Ray Society, Boston, Mass., Oct. 1–4, 1940.

process are more or less difficult to recognize. The more pathogenic species have lost their ability to grow by the sexual process and multiply in the tissue by asexual division.

Our series of cases of bronchomycosis consists of 79 cases, classified as follows: blastomycosis, 16 cases; actinomycosis, 10 cases; moniliasis, 40 cases; sporotrichosis, 3 cases; aspergillosis, 5 cases; geotrichosis and torulosis, 1 case.

berculosis in that the fibrosis is more of a network with heavy beading along these lines.

Coccidioidal granuloma has not been seen in our locality, except for 2 case histories and roentgenograms which were sent in to us. Carter,[1] of Los Angeles, found it the most common of the bronchomycoses. It was described by him as closely resembling tuberculosis, though the lesions may be cutaneous or oropharyngeal, with selective lymph node involvement. Blood vas-

Table I

INCIDENCE OF DISTRIBUTION OF THE MYCOSES

Type	Cases	Age in Years	Sex		Duration of Symptoms	Lung Involvement	Living Cases
			M	F			
Actinomycosis	10	8–66	8	2	7 wk. to 3 yr.	6	2
Blastomycosis	16	12–53	13	3	6 wk. to 3 yr.	10	3
Moniliasis	40	⅓–65	21	19	1 mo. to 5 yr. one 15 yr.	24	16
Aspergillosis	5	26–47	3	2	4 mo. to 1 yr.	3	3
Sporotrichosis	3	7–46		3	8 mo. to 8 yr.	1	1
Geotrichosis	2	25–25	1	1	2 to 3 yr.	1	1
Torulosis	1	25		1	6 mo.	1	0
*Coccidioidal granuloma	2	42	1			1	0

* Outside cases—Roentgenograms.

There was a slight predominance of the disease in the male: 47 males and 32 females. Classification according to age, sex, duration of symptoms, and survival is given in Table 1.

The roentgen appearance in the early stages resembles bronchitis or pneumonitis rather than tuberculosis,but as the fungi give rise to pus formation within the alveoli of the lung, there is either a resulting bronchiectasis or consolidating masses within the lung structure. In our series, the cases with *Monilia* infection seemed to present the most consistent roentgen changes. In the early stages, the lesions were diffuse, with small nodules throughout the entire lung field. In the later stages, the lungs presented a diffuse chronic interstitial fibrosis, extending from the hila throughout the lungs, with less involvement of the apices. This can usually be differentiated from tu-

cular dissemination seemed most common. Mediastinal adenopathy was present in 82 per cent of the cases.

Actinomyces is one of the more common fungi in this part of the South. About 60 per cent of our cases showed pulmonary involvement as well as skeletal changes. The pulmonary lesions are usually pneumonic in type with irregular areas of consolidation which often break down and suppurate, leaving large cavities. It often starts as a central pneumonic process and occasionally extends through the chest wall. Our case showing such lesions is now well nine years.

Wangensteen[13] gives a complete case report with review of the literature.

Blastomycosis, as described by Martin and Smith[6] and Doub,[2] produces characteristic skin lesions with thick, beefy proliferation. The lesions in the lungs are variable, from dense pneumonic masses to a

miliary distribution. One case, a young colored boy, had typical skin lesions, and also a diffuse nodular pulmonary involvement, simulating that often seen in moniliasis. Two of our cases are living, four and five years, free of disease.

Aspergillosis has been described fully by Sayers and Meriwether.[10] The roentgen appearance in their cases was more a miliary distribution. Our cases, however, showed more consolidation, simulating pneumonia. There was also pleural reaction in one case.

Geotrichosis has no typical roentgen appearance. According to Smith,[12] some strains are non-pathogenic to man. The lungs, when involved, resemble chronic tuberculosis with heavy studding along the bronchi; frequently cavitation occurs.

Sporotrichosis, as noted by Forbus,[4] seldom produces pulmonary lesions. One of our cases showed a diffuse nodular infiltration, radiating out along the bronchi in both lungs. The lesions have been reported, however, as being more often a dense gummatous mass with long mycelia invading the lung structure.

Torulosis is relatively uncommon in this locality, only one case being recorded. There is no typical roentgenological appearance. The lesions are often compatible with tuberculosis except that there is rarely cavitation.

Histoplasmosis, as described by Meleney,[8] should be mentioned as a number of cases have been described in various localities. The organism resembles a cryptococcus and pulmonary lesions are usually associated with liver and splenic enlargement. The lesions in the lungs may be both nodular and infiltrating or caseous with cavitation. There is no characteristic picture.

DISCUSSION

Bronchomycosis is either appearing more frequently in certain localities, or is being searched for more diligently. It should be considered one of the more common problems of differential diagnosis.

It is essential to remember that the mere presence of fungi in the sputum is not sufficient to establish a diagnosis of the disease. Many fungi in the sputum are not virulent and not pathogenic, whereas other fungi may be pathogenic but still not a cause of disease in the particular individual.

Roentgen findings are never typical and may closely resemble several pathological conditions. It is impossible to arrive at a correct interpretation of the roentgenograms of many of these patients suffering from bronchomycosis without the cooperation of the clinician and a pathologist who is an efficient mycologist.

The isolation of the fungi entails time and expense which as yet many institutions are not in a position to undertake, the presence or absence of tubercle bacilli in the sputum being all that is sought for.

The disease is not universally fatal, and some cases can be cured.

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DISCUSSION

Dr. Thomas R. Healy, Boston. I would like to congratulate Dr. Reeves on his excellent presentation.

I agree with Dr. Reeves that bronchomycosis is appearing more frequently in certain localities, but I believe it is due to more correct diagnosis.

In a report of *Monilia* infection of the lungs which I made in January, 1930, I pointed out that many cases of *Monilia* infection of the lungs were being overlooked, or diagnosed as tuberculous infections. This is not only shown by the greater number of cases being diagnosed in our laboratories and hospital services, but also by reports in current medical literature.

Generally speaking, the profession has thought of fungous disease as a tropical or semitropical disease, probably due to the fact that the maximum incidence occurs in warm, moist climates. However, cases are being reported in increasing numbers in all climates, but especially in our own. In my series of cases, only 20 per cent had ever visited the Tropics, and there were a few who had never been out of New England.

Yeast infection may occur at any age and in either sex, but in my series of cases (35 in number) the disease was found most often in the fourth and fifth age period, and generally in the male.

From the roentgenological aspect, the lungs show rather bizarre pathological changes, varying in degree from slight bronchitis in mild cases, to tubercle production, involvement of whole lobes with apparently parenchymal changes, or even cavitation, so that the picture may resemble tuberculosis, lobar pneumonia or bronchopneumonia.

Clinically, the signs and symptoms of a yeast infected chest vary with the extent and nature of the process. The clinical course extends from one to three months. Most of these patients recover spontaneously but some progress to the severe type of the disease, with a fatal termination. Observation leads me to believe that yeast infection in the lungs may be primary and not always secondary, as many claim. It is obvious that a diagnosis of yeast infection of the lungs cannot be positively diagnosed by roentgen examination alone. The picture may be that of almost any disease of the lung. However, the roentgenologist should always bear this condition in mind, especially in cases which show extensive lesions in the lungs, without correspondingly severe clinical signs and symptoms. And, as Dr. Reeves pointed out, without co-

COMPLICATIONS IN 16 CASES OF PULMONARY YEAST INFECTION

	Per cent		Cases	Per cent	
Pulmonary	69	Pneumonia	5	31	1 dead; pneumonia
		Pneumothorax	2	12.5	
		Tuberculosis	4	25	1 dead; miliary tuberculosis
Incidental	18	Syphilis	1		
		Hypertension	1		Dead; coronary thrombosis
		Atrophic arthritis	1		Improved after vaccine
None	13				

POSTMORTEM FINDINGS IN ONE CASE OF YEAST INFECTION

Pneumonia
Alveoli dilated
Cavitation
No tubercle bacilli found
Many yeast cells in cavities

END-RESULTS IN 16 CASES OF PULMONARY YEAST INFECTION

Well	7	
Improved at discharge	6	2 died later
Recurred after discharge	3	1 case recurred 3 times
Dead	3	All from other causes

operation of the clinician and the pathologist, it is impossible to arrive at a correct interpretation.

May I emphasize the care with which the sputum should be examined; the specimen must be of known bronchial origin.

I would like to present the statistics in my series of cases.

ADDITIONAL DISCUSSION*

DR. CARLETON B. PEIRCE, Montreal, Canada. Dr. Davenport has presented to us a subject in which I have been very much interested. I wish to take this opportunity to thank him for the reference to the work of Curtzwiler and myself.

In our experience, the roentgen treatment of tuberculous tracheobronchitis was not very satisfactory. However, those cases were not selected as he has suggested, and perhaps with proper selection of the case by means of bronchoscopy, confining the irradiation to only the "congestive" group, there may be some relief. This lesion, I believe, is far more frequent than we have believed up to this time.

I would like to suggest that those roentgenologists who have the opportunity to examine and treat this type of case should do so only in collaboration with a very experienced bronchoscopist, a thoracic surgeon, and an internist who are particularly competent in the treatment of pulmonary tuberculosis. Certainly, in the hands of the group with which I was associated, Dr. Alexander and Dr. Barnwell, and more recently Dr. Archibald, extreme caution had to be maintained in preventing complications in this particular type of tuberculous disease.

DR. LEO G. RIGLER, Minneapolis, Minn. I should like to ask Dr. Badger a few questions which may throw some light on problems that bother us a great deal.

The first refers to the gap in our knowlege which is gradually being filled in as to the significance of negative findings in the roentgenogram. Most of us can answer pretty well as to what value attaches to a positive finding, but what value attaches to a negative finding, is much more difficult.

Some years ago with Exner I published a paper in which we reported 5 cases studied in much the same manner as those Dr. Badger reported. These were all in medical students in whom we thought we could chronologically relate the exposure to tuberculosis with the occurrence of roentgen findings. We found that at least ten weeks would have to elapse from the time of exposure before any findings developed in the roentgenogram.

We have collected 6 more cases which seem to confirm that finding and I would like to know whether Dr. Badger has any information to offer which will help answer the question that we are asked so often: "What is the significance of a negative roentgenogram after esposure to tuberculosis and the occurrence of a positive tuberculin test"?

The second question relates to whether or not these lesions in the apex and in the subclavicular region are definitely what have been called first infection tuberculosis.

Has Dr. Badger any cases in which calcification has occurred in such lesions as it commonly does in children?

We have searched a very large number of cases with this in mind. I have sent for films all over the country on medical students and interns in the hope of getting films made early in the course of the lesion to determine whether or not calcification of these lesions will occur in the young adult. So far we have been able to find only three in which we could be reasonably certain.

I think it must be borne in mind that a negative tuberculin test is not complete evidence of the absence of a pre-existing lesion because the tuberculin test will change from negative to positive and from positive to negative under certain circumstances, so the fact that it was negative before does not, incontrovertibly at least, prove these lesions were first infections. I would like to hear comment on that.

DR. MERRILL C. SOSMAN, Boston. We are working on a series similar to Dr. Badger's in the Medical School at Harvard, and we are also trying very hard to find out whether it is a good thing to have a positive tuberculin test or a negative one. I hope Dr. Badger will tell us which is the best thing to have as you enter the school.

We have been disagreeably surprised by finding cases like Dr. Keefer has reported at autopsy, entirely unsuspected, or diagnosed entirely incorrectly, something that must certainly be kept in mind.

* Of papers by Drs. Badger and Ritvo; Keefer; Davenport; King, and Reeves.

Dr. Davenport, I think, has a particularly interesting group of cases which to me accounts for the rare case we have run across in the past, where a patient has a positive sputum and a negative roentgen finding. The tuberculosis may be limited to the trachea or the main bronchi, and it is the mechanical effect of obstruction which we must seek to avoid.

Going back through history a little bit, I might recall to your attention that collapse of the left lower lobe, as far as I know, was first discovered by Holmes and Churchill here in Boston, and was known for a while as the Holmes-Churchill line, i.e. the oblique line behind the heart at the left base. It was immediately forgotten, and has been rediscovered at least three times since then.

I call attention also to a paper by John Sproull, published some ten or twelve years ago, on acute pulmonary atelectasis due to tuberculosis, in all probability a case of tuberculous tracheobronchitis.

The sarcoid disease as presented by Dr. King and demonstrated by his roentgenograms is something I am sure almost all of us have overlooked or misnamed. In my own experience the fungous infections Dr. Reeves has reported have been entirely secondary infections; that is, they are saprophytic and rest on some other basic disease in our series and also in that of the Department of Pathology at the Harvard Medical School.

Carcinoma of the lung or bronchiectasis is the most frequent underlying condition when *Monilia* are abundant in the sputum. I doubt if the *Monilia* ever causes primary disease of the lung. Actinomycosis, blastomycosis and coccidioidal infection, however, may all be primary infections in the lung.

In respect to Dr. Badger's paper, I might add to the confusion by saying that we frequently find calcified hilar glands in our medical students with a negative tuberculin reaction. I am sure you have all had that experience.

Dr. Badger (closing). I don't think any final answer can be given to some of the points which Dr. Rigler has brought up. In the paper I have discussed some of these points.

In regard to the negative roentgenogram in the presence of a positive tuberculin test, I have shown that in the Boston City Hospital we do not feel there is any marked difference between the tuberculin positive and negative groups of individuals. We feel that for several reasons. In the first place, we have found almost the same number of lesions in both groups of cases. In the second place, some of the nurses and medical students must have received their infection plus their positive tuberculin test a very short time before they were admitted. Therefore, their condition is very similar to those who entered training with a negative tuberculin test, but who shortly after admission were exposed and became positive to tuberculin. The lag between the time of exposure and the development of a positive tuberculin test accounts for this.

We have found in those individuals we have been able to test frequently, that there is a lag of about ten weeks to three months between the beginning of exposure and the development of a positive tuberculin test. The shortest interval that we have had in our own series between the development of a positive tuberculin test and the appearance of a pulmonary lesion was a period of three months. The patient was roentgenographed every three months and did show a definite parenchymal change in three months.

I think we have to bear in mind that the tuberculin test must be done extremely frequently in order to establish the relation of the appearance of allergy to the time of infection and the appearance of an x-ray lesion. Dr. Esmond R. Long was here in Boston not long ago discussing this very matter. I was interested to know that he is also puzzled about the same question. I don't think the answer can be given. We haven't followed the cases for a long enough time, and the passage of time is most important in evaluating the pathogenesis of tuberculosis from its inception.

As to the appearance of calcium, we have not found it in any of our cases so far, but we have not completed the follow-up study. The cases which we have observed for the three year training period for nurses since 1932 will be followed up this year. So far, no calcium.

The second question was in regard to primary infections. Concerning Dr. Sosman's question about the medical students, I have also been interested in that same group with Dr. Eppinger. So far the series is relatively small, but the findings as we put them together last spring showed results very similar to the findings among nurses at the Boston City Hospital.

It didn't seem to make a great deal of differ-

ence whether the medical students entered training with a positive or negative tuberculin test. The amount of tuberculosis developed in the two groups has been closely similar. We feel that this study should be followed longer before we can answer definitely. Apparently there is some difference in the adult reaction to the primary infection as compared with the childhood reaction to the primary infection. Perhaps some of these adults have been infected previously in their childhood, the lesion has healed and become obsolete, their tuberculin test has become negative again, and they are ready for another primary infection.

I have been looking for that group but so far have not had any who can be so classed. We do not know whether those positive to tuberculin in childhood who become negative to tuberculin later have a subsequent primary reaction which is different from the initial primary reaction. Evidence is accumulating to show that the adult response to a primary infection *is* different from the childhood response. But to what factors this difference is due there appears to be no answer at present.

I don't think we know definitely whether or not a positive tuberculin test offers a greater degree of safety to those who are exposed to tuberculosis. We do know by the natural laws of survival that those who have their primary infection and do not develop clinical tuberculosis must have a degree of constitutional resistance to tuberculosis that is greater than those who develop the disease. Time remains an important element in evaluating individual resistance to tuberculosis. The tuberculin test should not distort our perspective.

Dr. Keefer (closing). I want to thank Dr. Butler, Dr. Portmann, and Dr. Sosman for their very generous remarks concerning the paper which I presented this morning.

In answer to the question as to whether or not the infection of the tracheobronchial lymph nodes is secondary to a previous infection in the lung, that tuberculosis in lymphoid tissue may occur as a primary lesion I don't think there is any doubt. The tonsil is an excellent example of that. On the other hand, when you study the tuberculosis of the tracheobronchial lymph nodes, it is very difficult to say that this infection can arise as a primary infection without preceding infection of the lung. Certainly in many patients who have tuberculosis of the tracheobronchial lymph nodes, one cannot demonstrate at the time parenchymal lesion of the lung, and this is especially true in adults.

It would be my general impression at the moment that tracheobronchial lymph node tuberculosis in adults without a demonstrable parenchymal lesion was probably either a childhood form of reaction or a reactivation of a previous focus of tuberculosis in the lymph nodes.

Dr. Davenport (closing). I should like to make it perfectly clear, which apparently I did not, that we are not advocating the radiation treatment in all tuberculosis. We are advocating irradiation only to control the acute congestive phase of this disease. It is perfectly obvious that the chronic cicatricial stenosis cannot be treated. The damage is done and the airway is narrowed. By far the greater majority of advanced cases of pulmonary tuberculosis show bronchial ulcerations but give no symptoms. The ulceration is associated with a parenchymal disease, and the prognosis of this condition in that particular group of cases is dependent on the parenchymal disease in the lung.

It is in a group of small cases only, where the manifestations are largely those of obstruction with fulminating symptoms due to retained secretion, that we are advocating this type of treatment, and while we have found at postmortem that more than 50 per cent show discrete ulcerations, we are not attempting to treat those ulcerations.

Bronchoscopically in five years we have found less than 4 per cent have the acute fulminating disease where the primary involvement was in a bronchus and the patients have died of obstruction. We are trying to control the lymphocytic obstruction and engorgement if feasible.

The question was asked whether the original focus may be in the lymph nodes. I don't know, but I think we can safely say that the lymph node enlargement and the lymphocytic engorgement of bronchial mucosa is a predominant picture here, and it is in that particular type of lesion where we may expect radiation therapy to be of some benefit.

I would agree with Dr. Peirce that if you treat all cases of ulceration of the bronchial mucosa, including those that already have advanced parenchymal disease, the results will be

very confusing, because it is impossible to evaluate the prognosis in the one case as due to the bronchial disease, or in the other as due to the associated parenchymal lesions. We are treating, then, only those cases where the primary manifestations and where the acute clinical symptoms are due to congestion and engorgement of the bronchial mucosa.

Dr. Reeves (closing). I agree with Dr. Healy that the fact that more cases of bronchomycosis are being found each year may not mean it is increasing but it may be that we are more aware of the fact and more studies are being made, and more repeated studies are being made.

Dr. Sosman mentioned the fact that *Monilia* is frequently found in concurrent diseases as a saprophyte. I think that is quite true. Especially in carcinoma of the lung it is very frequently found as a secondary agent, but, as I mentioned previously, there are many carriers who also have pathogenic *Monilia* in their sputum but still show no pulmonary infections themselves, whereas other patients, we think, have a primary pulmonary infection of the lungs.

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LEFT VENTRICULAR ANEURYSM

REPORT OF CASE

By W. C. HUYLER, M.D.
Roentgenologist, The Mutual Life Insurance Company of New York
NEW YORK, NEW YORK

IN THE work in this laboratory, which consists largely of reviewing roentgenograms submitted from various sources, we receive so many studies of the heart which include only a single posteroanterior view that we are persuaded that the value of oblique views is not widely enough recognized. There is a large number of borderline heart cases in which the single posteroanterior view leaves a question as to the size of the heart or its individual chambers. We feel that it is not wise to diagnose a borderline enlargement without corroborative evidence from an oblique view. It is our practice here to take posteroanterior and left anterior oblique views on every heart case, and a right anterior oblique view if it is indicated by the roentgenoscopic study—which also should be done on every case. That it is possible to completely miss important pathological conditions in a single posteroanterior view is demonstrated by the case reported below, in which only the oblique view shows the condition.

CASE REPORT

This case is that of a fur merchant, aged fifty-six, whose illness began with a sudden attack of precordial pain radiating to the back and to both arms while riding in a railroad train in December, 1934. For ten days after this he had an afternoon temperature up to 101° and 102° F.; blood pressure was 105/60; and pulse was 90. Since this time there has been definite precordial pain on effort, radiating to the left arm. We have only rather brief records of the period from the onset to May, 1939. These indicate that the blood pressure remained consistently low, one reading being 90/60; the pulse ran between 80 and 90. An electrocardiogram made shortly after onset showed a Q-R-S of .14, slurring and low voltage in all leads, T inverted in lead I, and a deep Q in lead III. In July, 1936, the Q-R-S was found to be .14. In July, 1937, the patient was complaining of fatigue and dyspnea in addition to his anginal pain. Four months later the heart sounds were found to be of poor quality, and the electrocardiogram is reported as showing all R waves

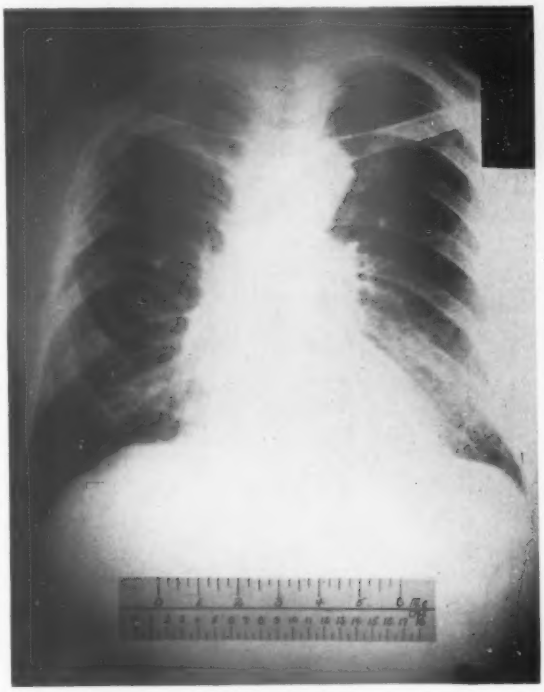

FIG. 1

slurred, with the R-S in lead IV of the "M" type and slurred. Roentgenoscopic examination at this time is said to have been negative. In April, 1939, the heart sounds were again reported to be of poor quality and there was a short systolic murmur at the apex. Roentgenoscopic examination at this time is said to have shown the heart moderately enlarged and the aorta markedly widened. On May 16, 1939, the patient was complaining of constant substernal pain, with precordial pain radiating to the left arm on any effort. Examination showed a rough systolic murmur at the apex and in the

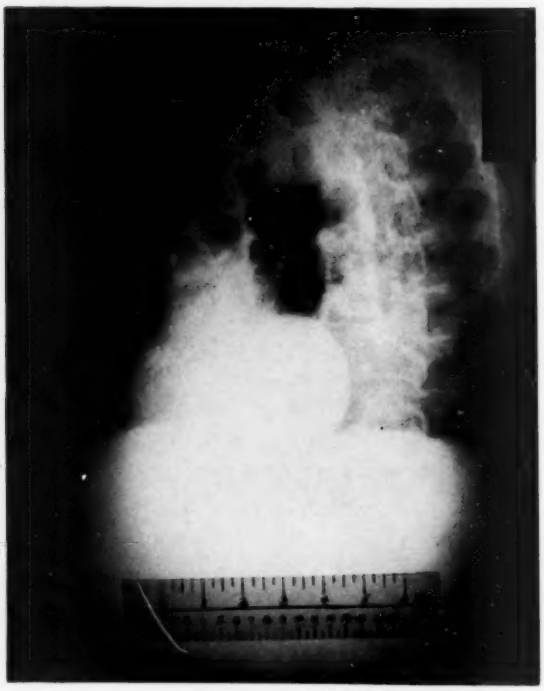

Fig. 2

aortic area, without palpable thrill. Blood pressure at this time was 130/90, with pulse 80. The electrocardiogram showed sinus arrhythmia, rate 69, P-R interval .14, Q-R-S duration .13, left axis deviation modified by deep inspiration, Q-R-S slurred in leads I, II and III and bizarre in lead IV, diphasic T in lead IV. Posteroanterior and left anterior oblique roentgenograms of the chest were made, and it is in these that the chief interest in this case lies.

The posteroanterior roentgenogram of the chest (Fig. 1) shows evidence of healed primary tuberculosis and a moderate general accentuation of lung markings involving particularly the secondary trunks and especially marked at the bases. In this view the heart is entirely normal in size, shape and position. The aorta is long and tortuous and somewhat denser than normal, with the knob outline accentuated. There is no widening of the aorta. In the left anterior oblique view, the posterior surface of the left ventricle bulges markedly, as shown in Figure 2. This is considered to be a posterior left ventricular aneurysm, the presence of which cannot be demonstrated in the posteroanterior view.

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CALCIFICATION OF THE CHOROID PLEXUS AND ITS DISPLACEMENT BY EXPANDING INTRACRANIAL LESIONS*

By A. E. CHILDE, M.D.
MONTREAL, QUEBEC

STEREOSCOPIC roentgenograms of the skull frequently reveal intracranial calcification. Calcium is visible in the pineal body in more than 50 per cent of adult skulls, and it is often seen in the falx and in the tentorium. So far as is known at the present time, the presence of calcium in these structures is normal or at least has no clinical significance. There is considerable literature dealing with displacement of the pineal body in various types of expanding intracranial lesions. Calcification also occurs in the choroid plexus of the lateral ventricles. Heuer and Dandy[5] were able to verify by autopsy that calcification shown in roentegnograms was situated in the choroid plexus. Sachs and Whitney[9] reported the case of a microcephalic idiot who presented marked calcification in each choroid plexus. Camp[2] stated that calcium is demonstrable in the choroid plexus relatively often but is probably frequently misinterpreted. Beals[1] in a selected series of 86 cases over twenty years of age found calcification in 28 per cent and considered it to lie in the glomus. Dyke[3] was able to recognize calcium in the choroid plexus in 5.1 per cent of a series of 2,724 roentgenograms of the skull. Dyke and Davidoff[4] pointed out the defect produced by the choroid glomus in encephalograms and showed that calcification sometimes occurred within this shadow.

An analysis of 1,000 consecutive skull examinations made at this institution revealed calcification to be present in the cho-

TABLE I

CALCIFICATION IN CHOROID PLEXUS AS SHOWN BY SKULL ROENTGENOGRAMS OF 1000 CONSECUTIVE PATIENTS

Age	0–10		11–20		21–30		31–40		41–50		51–60		61–70		71–80		Over 80	
Sex	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F
Number of Patients	63	51	80	60	80	68	120	68	106	66	80	48	52	34	12	10	2	0
Calcification in Choroid Plexus	0	0	0	2	4	3	16	4	16	7	22	6	17	5	5	4	1	0
Percentage Calcification — Male and Female	0	0	0	3.3	5	4.4	13.3	5.9	15.1	10.6	27.5	12.5	32.7	14.7	41.7	40	50	0
Total Percentage of Calcification	0		1.4		4.7		10.6		13.4		21.9		25.6		40.9		50	

Total incidence 11.2%
Incidence over 20 years 14.7%
Total male incidence 13.6%
Total female incidence 7.65%

Bilateral calcification—choroid plexus 7.4%
Calcification on right side 1.9%
Calcification on left side 1.9%

* From the Department of Neurology and Neurosurgery, McGill University, and the Montreal Neurological Institute, Montreal.

roid plexus in 112 cases, or 11.2 per cent (Table 1). In 7.4 per cent the calcification was bilateral; it was situated on the right side only in 1.9 per cent and on the left side only in 1.9 per cent. The incidence of calcification increased in direct proportion to the age. No such calcification was encountered in the group before the age of ten years, and it was observed in only 1.4 per cent in the eleven to twenty year group. In those patients over twenty years the total average incidence was 14.7 per cent, and, in the seventy-one to eighty group it had risen to 40.9 per cent. It is also interesting to note that it occurred almost twice as frequently in males as in females—13.6 and 7.65 per cent respectively.

The location of such calcification almost always corresponds to the position of the glomus as may be easily proved by encephalography or ventriculography.

Very occasionally calcification occurs elsewhere in the ventricle, apparently in the choroid plexus, usually at some point between the glomus and the foramen of Monro. This type of calcification tends to be linear and usually does not resemble calcification situated in the glomus.

The glomus is that portion of the choroid plexus situated in the lateral ventricle where the choroid bends upon itself in its course from the temporal horn to the foramen of Monro. In this region the plexus becomes expanded. It lies in intimate contact with the anterior ventricular margin and is usually situated slightly above the level of the occipital horn, or, calcar avis, if the occipital horn is not present. In lateral roentgenograms, calcification in the glomus is situated a short distance above and posterior to the position of the pineal, well away from the midline. In anteroposterior or posteroanterior roentgenograms, it is situated 2.5 to 3 cm. to either side of the midline, usually slightly closer to this plane than to the inner table of the skull. Such calcified areas vary in size from minute specks, which are seen with considerable difficulty, to large dense areas occasionally over 1 cm. in diameter. They may be rounded or not uncommonly kidney-shaped being convex posteriorly and slightly concave anteriorly. This shape may be so characteristic that even when unilateral calcification is present, the appearance is quite typical.

Very little has been written concerning displacement of the calcified glomus, as shown in plain roentgenograms. Newell[8] mentioned an instance in which one choroid plexus was displaced downward by pressure from a cerebral tumor. Lowman and von Storch[6] described displacement and recession of the choroid plexus by what they thought to be a localized cerebral hemorrhage, but they did not produce operative or autopsy confirmation as to the site or type of lesion.

Eight cases have been encountered in this hospital in the course of twenty-one months in which there was evident displacement of a calcified glomus. In 7 instances the significance of the displacement was suspected before pneumography and operation, but in the eighth case the calcified glomus was originally thought to represent a partially calcified neoplasm, and the correct interpretation was not arrived at until after ventriculography. During the same period of time there has been no instance in which definite displacement was suspected and later proved unimportant.

The following case reports are listed in chronological order together with the most important available data. The diagrams of the skull and calcified intracranial areas are reproductions of pantograph tracings of the roentgenograms. Pneumographic analysis charts are included, prepared in the same manner as in previous publications.[7] The first row of figures in these charts shows normal ventricular outline in diagrammatic form, the second row the ventricular tracings from the case in question and the third the size and position of the expanding lesion as plotted by the neurosurgeon.

REPORT OF CASES

Case 1. G. B., male, aged forty-two, was admitted November 30, 1937. Unconsciousness and drowsy periods had occurred following a head injury nine days previously, sustained in a fall from the top of a railroad tender to the tracks beneath. On admission there were found to be marked drowsiness, complete paralysis of the right face, of the central type, of the right arm and apparently of both legs. The deep reflexes were diminished to absent and the superficial reflexes were absent. Both pupils were small, the left being slightly larger than the right, and the left reacting sluggishly to light. There was nystagmus with quick phase to the left on attempt to gaze to the left. A subcutaneous hematoma was present about the left eye and in front of the left ear as well as a small hematoma in the left parieto-occipital region. Bleeding had occurred into the right external auditory canal and behind the right tympanic membrane. There was also slight bleeding behind the left tympanic membrane.

Examination of the spinal fluid showed the initial pressure to be 200. The fluid was clear yellow and it contained 350 red blood cells, 10 white blood cells; the Pandy was ++ and proteins 208 mg. per 100 cc.

Roentgenographic examination revealed multiple fractures. One passed through the middle third of the shaft of the right tibia. There was a compression fracture involving the body of the fifth cervical; the transverse processes of the right side of the third and fourth lumbar were detached. Roentgenograms of the skull showed one linear fracture commencing near the right mastoid and running directly upward to cross over to the left side into the left parietal bone. There was no depression of these fragments. A second linear fracture was present running horizontally through the left parietal bone, about half way between the base and the vertex, and extending forward into the left side of the frontal bone. This latter fracture was associated with slight depression. There was a third fracture running downward toward the left mastoid. The pineal body was calcified and situated 6 mm. to the left of the midline. There was calcification apparently in the glomus of each choroid plexus. The calcification on the right side was situated 1 cm. anterior to that on the left and it was also 1.6 cm. closer to the midline (Fig. 1, *A* and *B*). It was felt that a collection of blood was present in the right side of the skull well posteriorly.

Bilateral exploration was carried out later on the same day. On the left side some slight depression of the fragments was found but there was no evidence of extradural or subdural bleeding. Hemorrhage had occurred into the leptomeninges and there was evidence of petechial hemorrhages in the cortex but no disintegration. On the right side an extradural hemorrhage 3 cm. thick covered the posterior part of the parietal lobe and the occipital lobe. There was also a moderate quantity of subdural fluid but the leptomeninges were free from blood and the underlying cortex was not abnormal. The brain

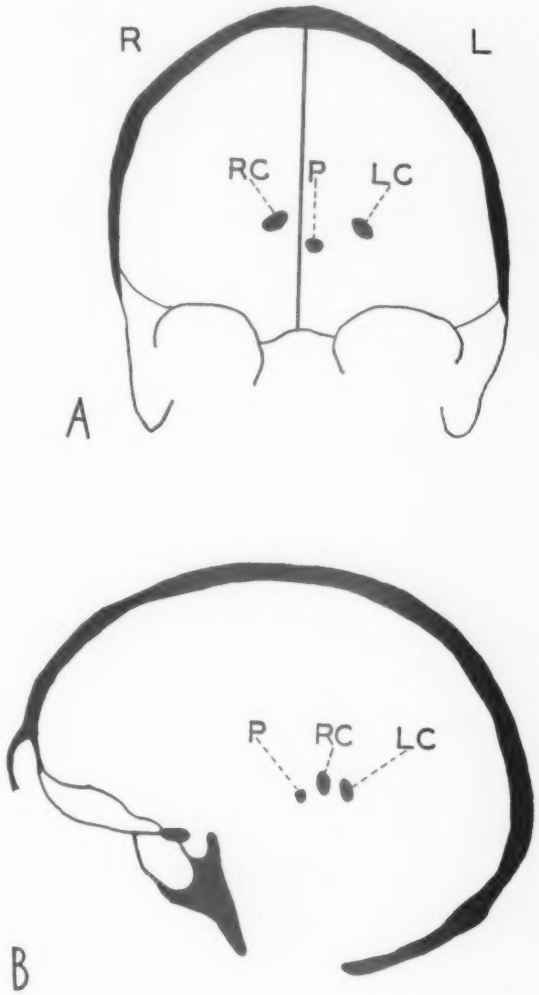

Fig. 1, *A* and *B*. Case 1. Anterior and medial displacement of calcification in the glomus of the right choroid plexus produced by a large extradural hemorrhage covering the posterior part of the parietal lobe and also the occipital lobe. *RC*, right choroid; *LC*, left choroid; *P*, pineal.

expanded following removal of the extradural clot.

Case II. J. C., male, aged sixty, was admitted March 17, 1938. He complained of diminishing visual acuity for the preceding six to eight weeks with a marked tendency to run into objects, particularly those approaching from the right side. He also complained of occipital headaches, increasing in severity, slightly worse on the left, which had been present for the same period of time. There had been coincident amnesic aphasia. Examination revealed diminished visual acuity (left 3/23; right 3/33), definite full right hemianopsia, fullness of retinal veins and papilledema, particularly on the right. The right side of the face was weak and the palate arched to the right on phonation. There were absent left abdominal and cremasteric reflexes, diminished tendon reflexes in the right leg, absent position sense in the right leg, almost absent two-point discrimination in the right hand and foot, and astereognosis of the right hand; the finger-nose-finger and finger-finger tests showed diminished position sense in the right arm with a tendency to drift to the left; the right hand wandered to the left when the head was rotated to the left and there was past-pointing on the right.

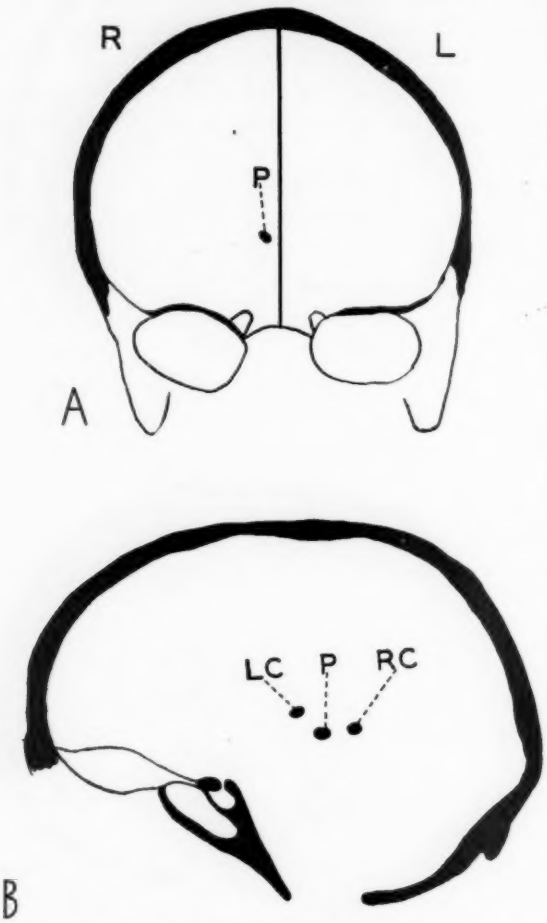

Fig. 2, *A* and *B*. Case II. Calcification in the left choroid plexus is displaced anteriorly by a glioblastoma multiforme deep in the left parietal lobe.

Roentgenographic examination of the skull on the day of admission showed slight atrophy of the dorsum sellae and posterior clinoids. Linear calcification was visible to either side of the sella, presumably in the internal carotids. The pineal body was calcified and situated 8 mm. to the right of the midline. There was calcification considered to be in the glomus of each choroid plexus, only visible in the lateral films. The situation seemed normal on the right side but on the left the calcification was approximately 2.5 cm. farther anteriorly (Fig. 2, *A* and *B*). The roentgenographic impression was an expanding lesion located in the left posterior parietal or occipital lobes. At operation, a single burr hole was made over the left parietal eminence about 3 cm. from the midline. An exploring brain needle met resistance at a depth of about 2 cm. and a brain worm was obtained which showed infiltrated brain and frank necrotic tumor. Microscopic examination revealed a glioblastoma multiforme. No further operative maneuvers were attempted but the patient was given deep roentgen therapy.

Case III. G. C., male, aged sixty-six, was admitted May 9, 1938. The complaints were weakness and absent sense of smell for the previous year, occipital and temporal headache, increasing drowsiness and left hemiparesis. The findings on admission were marked drowsiness, congestion of retinal vessels with blurring of disc margins, enlargement of the left pupil in comparison to the right, and sluggish pupillary reaction. There were also left-sided paralysis, left facial weakness, increased left tendon reflexes, diminished left cremasterics, left plantar extension, diminished sensation of the left side of the body including the face and questionable left hemianopsia.

Roentgenograms of the skull on May 9, 1938, revealed no abnormality of the sella turcica. Pineal calcification was only visible laterally and no displacement was shown. Faint calcification was present in the region of the left choroid

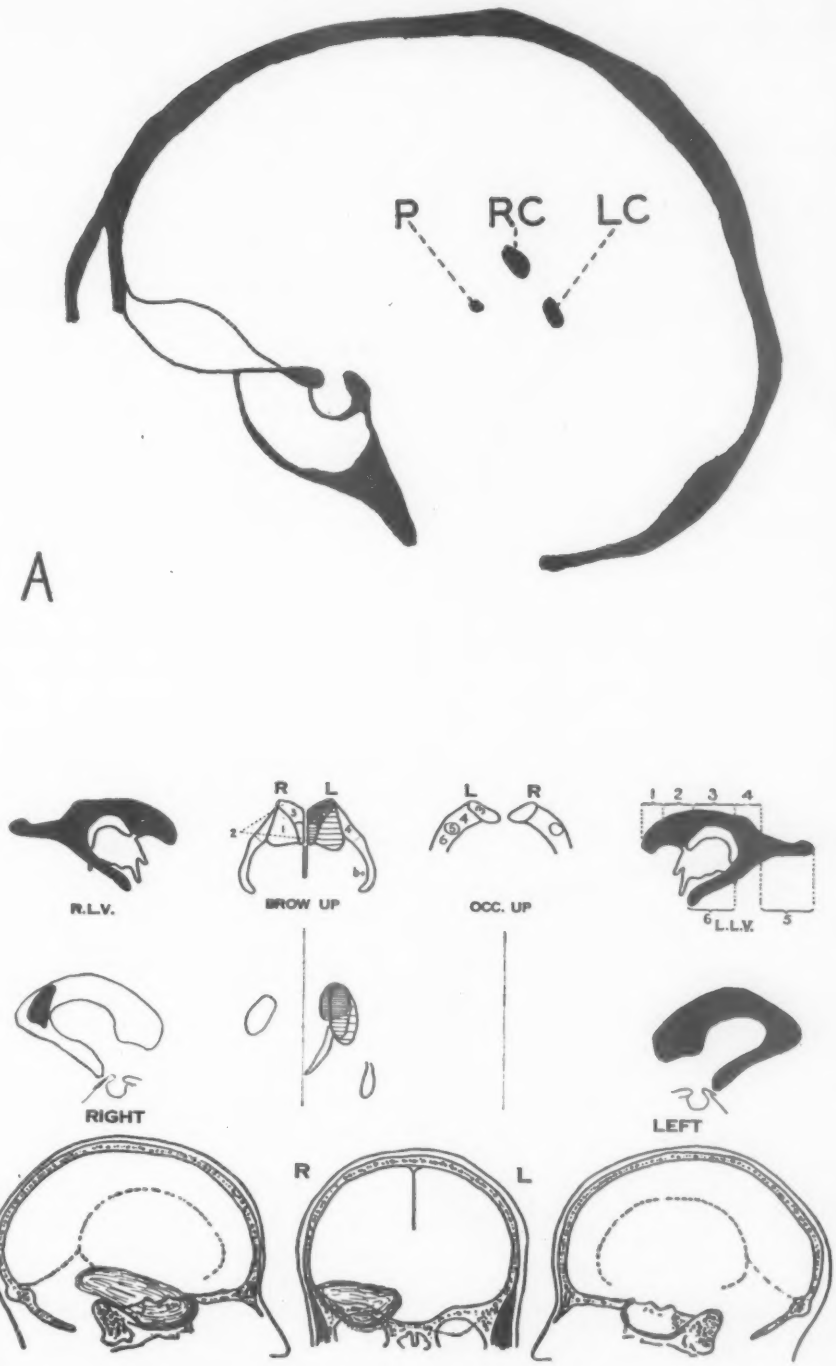

Fig. 3, *A* and *B*. Case III. Anterior and upward displacement of calcification in the right choroid plexus, resulting from a glioblastoma multiforme in the right temporal lobe. This tumor extended medially almost as far as the midline.

glomus. There was more marked calcification considered to be in the glomus of the choroid plexus of the right lateral ventricle, and this was situated 1.8 cm. higher than on the opposite side as well as 0.8 cm. anterior (Fig. 3, *A*). The roentgenographic impression was an expanding lesion involving the right cerebrum, extending deeply inward so that it elevated the posterior

portion of the right lateral ventricle. Ventriculography on May 10, 1938, showed extremely marked displacement of the ventricular system from right to left. The left lateral ventricle was only slightly enlarged. Portion 4 of the right lateral ventricle was pushed upward and forward (Fig. 3, *B*). At operation on the same day a glioblastoma multiforme was found to present on

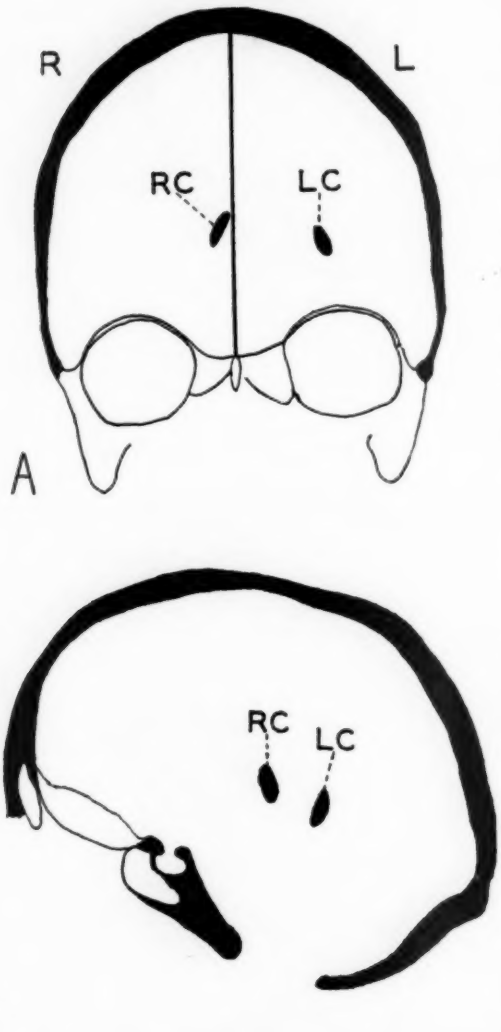

FIG. 4, *A* and *B*. Case IV. Marked medial and anterior displacement of the right choroid glomus, produced by a glioblastoma multiforme situated in the right parieto-occipital region.

the surface of the right temporal lobe below the sylvian fissure. A very large portion of this lobe was removed, the removal being carried down to the midline exposing the free edge of the tentorium of the cerebellum.

CASE IV. C. B., male, aged sixty-one, was admitted on August 6, 1938, complaining of headache for three weeks, tinnitus for two weeks and dizziness and stumbling gait for one week. He had fallen downstairs three days before admission. Examination revealed a left homonymous hemianopsia and papilledema. There were slight weakness and diminished coordination in routine tests of the left arm and leg.

Roentgenograms of the skull revealed no deformity of the sella turcica. The pineal was not seen in the anteroposterior roentgenograms and very questionably in the lateral films. There was calcification considered to be in the glomus of each choroid plexus. This could be recognized not only in lateral but also in anteroposterior stereoroentgenograms. The calcification on the right side was displaced medially to within 0.3 cm. of the midline, slightly upward and 2.3 cm. forward (Fig. 4, *A* and *B*). The roentgenographic impression was a very large expanding lesion situated posteriorly in the right hemisphere displacing the glomus of the right choroid plexus markedly medially and forward as well as slightly upward. Craniotomy in the right parieto-occipital region was performed two days later. A cyst from which clear yellow fluid escaped overlay a typical glioblastoma multiforme. The bulk of the tumor was removed by the electrocautery and suction, and during this procedure the body of the right lateral ventricle was entered. Re-examination of the skull twelve days later showed that the calcified shadow on the right side was no longer present.

CASE V. R. S., male, aged forty, was admitted September 16, 1938. The main complaint was weakness of the right side of the body including the lower arm and the right face, but he also suffered from left-sided headache, generalized pain, sleeplessness and increased worry. The findings were dilated pupils, bilateral papilledema with engorgement and tortuosity of the retinal veins and right homonymous hemianopsia. There were questionable left sixth nerve paresis, lower facial weakness on the right, slight deviation of tongue to right, weakness of right arm and leg with spasticity, hyperactive deep reflexes on the right, absent abdominal and cremasteric reflexes on right, positive Hoffmann on right and diminished position sense of toes on right.

Roentgenograms of the skull showed no deformity of the sella. The pineal body was not calcified. On the left side faint linear streaks of

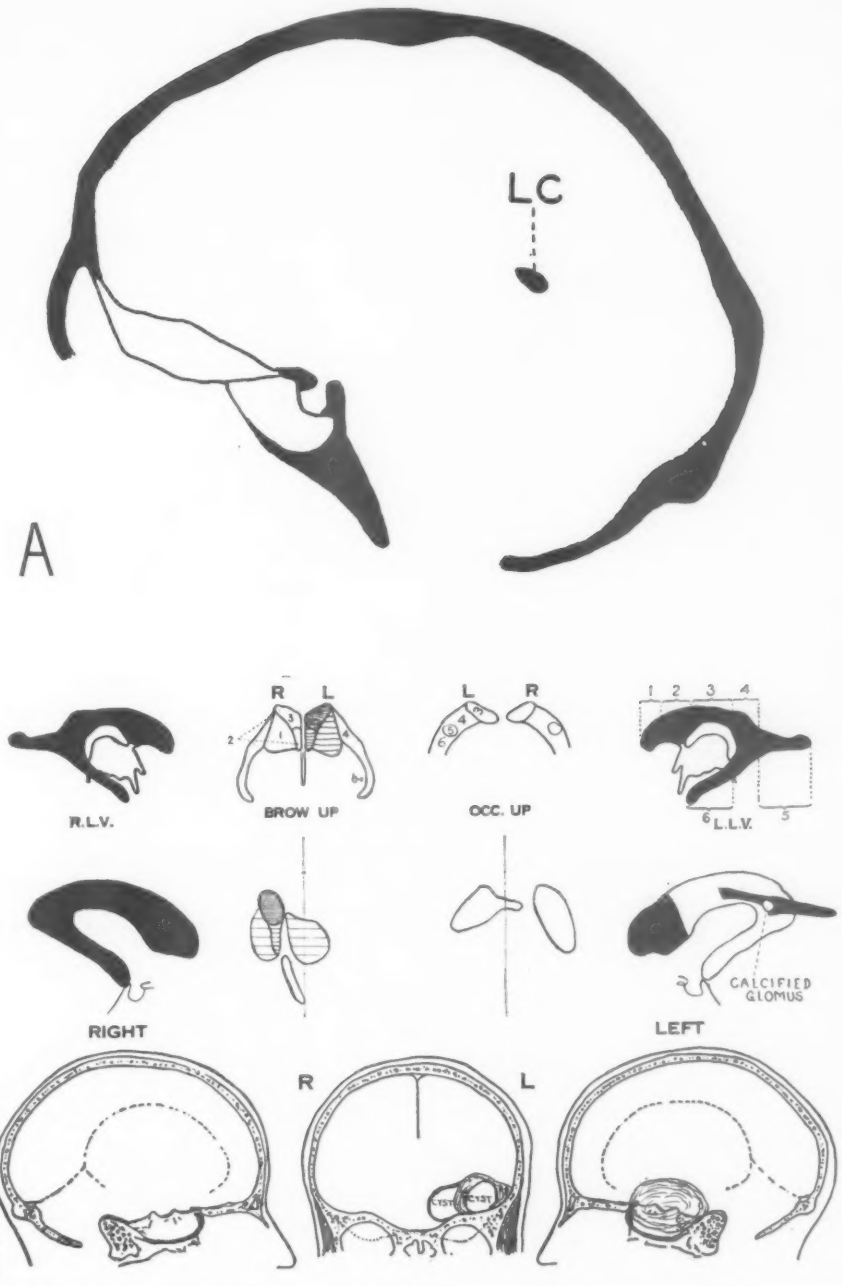

Fig. 5, *A* and *B*. Case v. Posterior displacement of a unilaterally calcified choroid glomus resulting from a metastatic carcinoma situated in the posterior portion of the left temporal lobe. The calcification was originally thought to indicate the site of a tumor.

calcium were distinctly visible somewhat posterior to the position usually occupied by calcification in the glomus of the choroid plexus (Fig. 5, *A*). It was felt that these probably represented the site of a neoplasm.

Ventriculography on September 22, 1938, showed the septum pellucidum to be displaced 1.3 cm. to the right of the midline. The right lateral ventricle was moderately dilated. The left lateral ventricle was markedly compressed from above and slightly from below in portion 3, and this portion ran obliquely from left to right. Portion 4 was compressed from above and below and its anterior border was displaced pos-

teriorly. Portion 5 was thin and slightly elevated. There was no visualization of portion 6 (Fig. 5, *B*). The calcification seen in the flat films was shown to be in the left lateral ventricle and there seemed to be no doubt that it was situated in the glomus of the left choroid plexus.

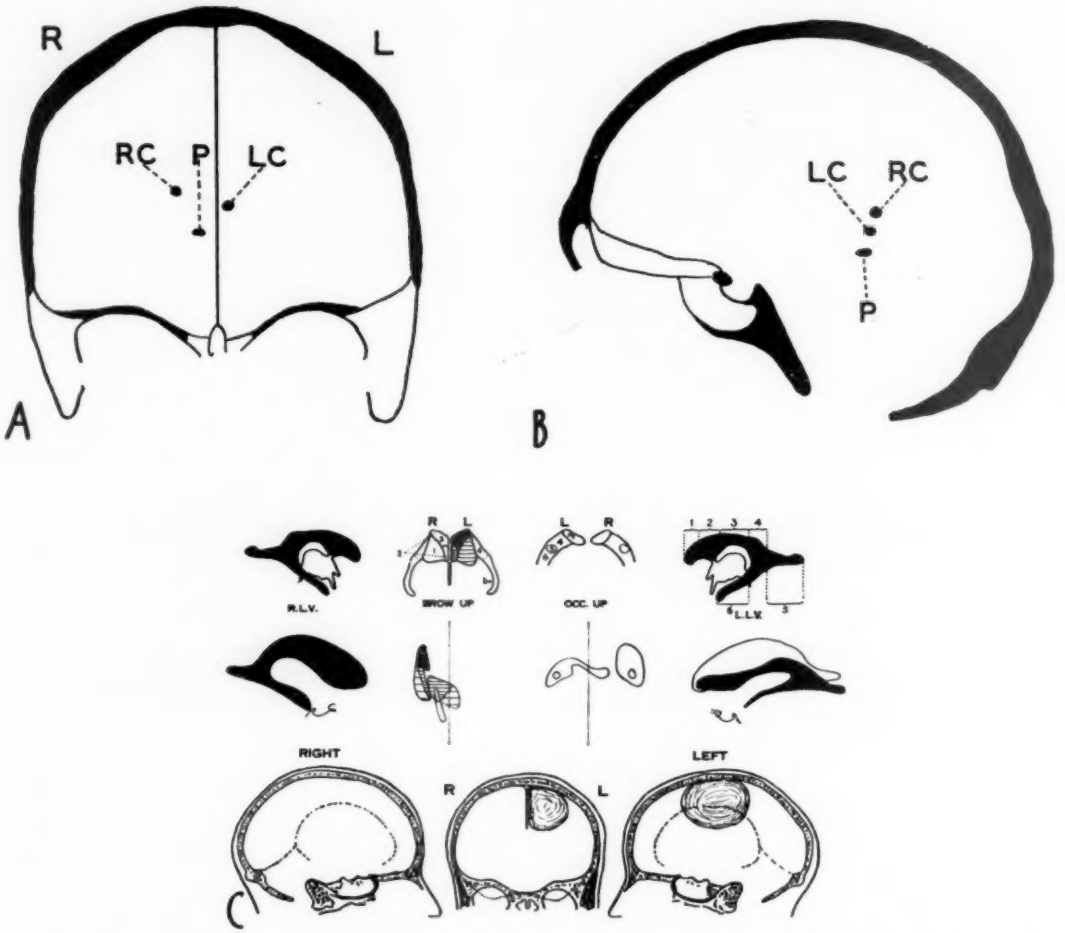

Fig. 6, *A*, *B* and *C*. Case vi. Medial and downward displacement of calcification in the left choroid glomus resulting from a meningeal fibroblastoma arising from the lateral edge of the median longitudinal sinus.

The choroid plexus was displaced posteriorly and slightly upward; the calcium did not lie in a neoplasm, as was originally suspected. Examination of biopsy material removed at the time of ventriculography from the posterior portion of the left temporal lobe revealed metastatic carcinoma.

Case vi. J. P., male, aged fifty-four, was admitted on March 13, 1939. The entrance complaints were difficulty in speech and weakness of right limbs for two years, worse in the previous ten months. Diplopia had been present for three weeks. There had been generalized headache lasting two or three hours every three days for two weeks. Examination revealed a moderate degree of motor aphasia with difficulty in finding words, bilateral papilledema, deviation of the uvula to the left on phonation and diminution of power in the right upper extremity without increase in tone. There were positive Babinski and Hoffmann reflexes on the right and increased tendon jerks of the right upper extremity.

Roentgenographic examination of the skull revealed marked erosion of the posterior clinoids and dorsum sellae. The pineal was faintly calcified, somewhat posterior to the normal zone and 7 mm. to the right of the midline. There was also calcification more readily recognized in the anteroposterior roentgenograms considered to

be in the glomus of each choroid plexus. On the left side it was situated 0.8 cm. lower than on the right and also 1.2 cm. closer to the midline (Fig. 6, *A* and *B*). The impression was a large left-sided expanding intracranial lesion growing moderately slowly, situated either in the temporal, posterior frontal or anterior parietal regions. A ventriculogram on March 15, 1939, showed the septum pellucidum to be displaced slightly over 2 cm. to the right of the midline. Portions 1, 2 and 3 of the left lateral ventricle were displaced to the right, downward and compressed markedly from above, the maximum deformity corresponding to portion 3. There was also some compression from above and depression of portions 4 and 5, and portion 6 was flattened (Fig. 6, *C*). The diagnosis was a left-sided expanding lesion situated predominantly in the posterior portion of the frontal lobe quite high up in the skull. Operation uncovered a typical meningeal fibroblastoma arising from the lateral edge of the median longitudinal sinus, rather lightly attached to the adjacent dura and falx.

Case vii. F. C., male, aged fifty-five, was admitted April 5, 1939. The complaints were convulsive seizures which had occurred on December 1 and 21, 1938, and February 14, 1939. There had been an attack of dizziness and marked stiffness on March 15, 1939, during which the patient could not talk but did not lose consciousness. This was followed by diplopia and incoherence for thirty minutes. He had been unable to read for three months. The past history was unimportant except for a head injury ten years before admission with uncon-

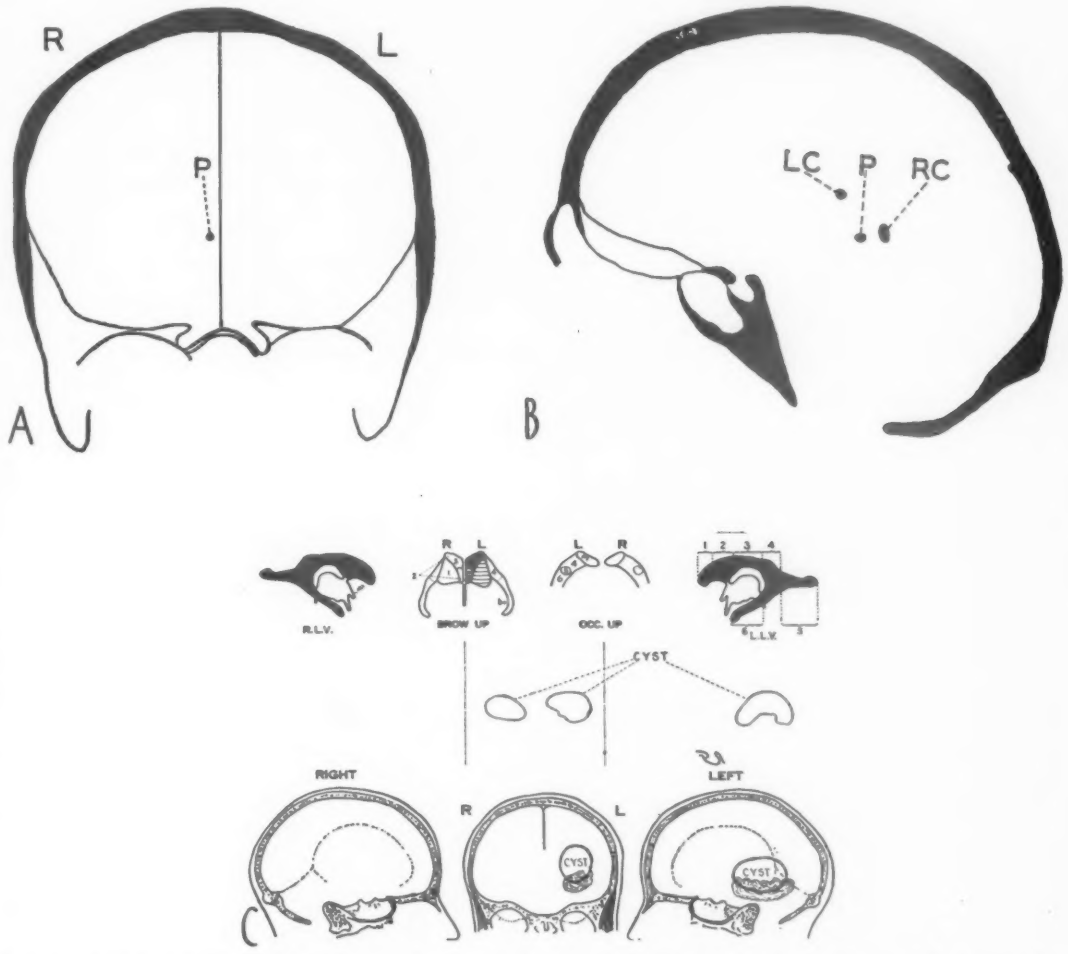

Fig. 7, *A*, *B* and *C*. Case vii. Anterior and upward displacement of the calcified left choroid glomus, due to a cystic glioblastoma multiforme situated deep in the posterior temporal and parietal lobes.

sciousness for an indefinite period. The findings showed a nominal aphasia with perseveration, apparent lack of concentration, early bilateral papilledema, right homonymous hemianopsia, absent superficial reflexes, right side, diminution of deep reflexes of lower extremities, coarse tremor of right hand on voluntary movement and diminution of auditory acuity.

Roentgenograms of the skull revealed no definite abnormality of the sella turcica. There were two small areas of calcification considered to be in the pineal body and these were displaced 6 mm. to the right of the midline. The lateral films showed calcification in the position of the glomus of the right choroid plexus. Another calcified area was visible on the left side 1.9 cm. anterior to and 1.6 cm. above the position of the glomus of the choroid plexus on the right side (Fig. 7, *A* and *B*). The impression from the plain films was a rather rapidly growing expanding left-sided intracranial lesion, very possibly situated in the occipital or parietal lobes. Electroencephalography suggested a diffusely infiltrating and expanding lesion in the left temporoparietal region, causing extensive injury to the brain in this locality. Operation on April 6, 1939, revealed at a depth of 4 cm. a cyst containing clear yellow fluid which coagulated almost immediately. This overlay a solid neoplasm which proved to be a glioblastoma multiforme deep in the posterior temporal and parietal lobes (Fig. 7, *C*).

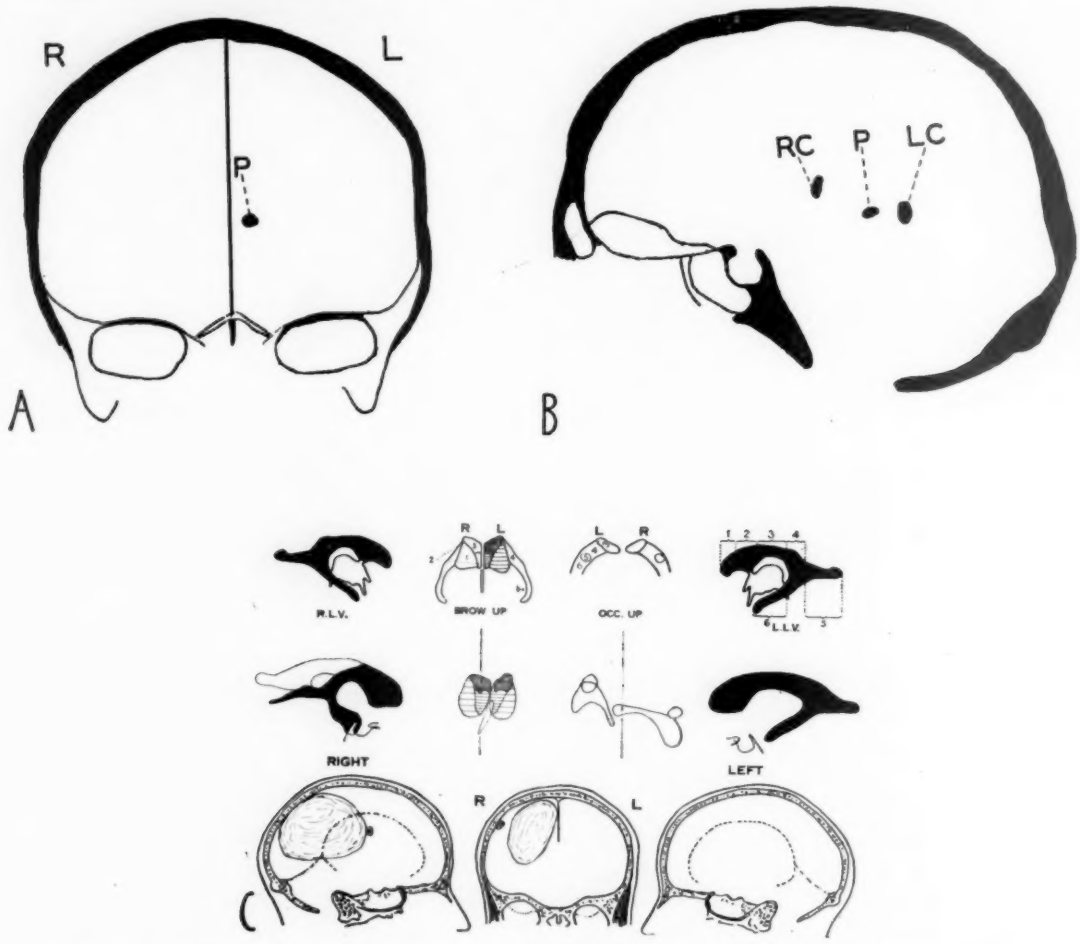

Fig. 8, *A*, *B* and *C*. Case viii. Pronounced anterior displacement of the right choroid glomus resulting from a meningeal fibroblastoma adherent to the posterior part of the falx.

Case viii. L. M., female, aged sixty-two, was admitted August 8, 1939. She complained that she had been unable to see objects on the left side with the left eye, for three years. Her memory had deteriorated. There had been nausea

without vomiting for one year and a dull frontal headache and aching pains in the leg and back of the neck for six months. She was dizzy when lying in bed or when she suddenly changed her position, and general weakness especially of the left arm had been present for the preceding few months.

and displaced 1 cm. to the left of the midline. There was calcification in the usual position of the glomus of the left choroid plexus. The same type of calcification was also visible deep in the right cerebral hemisphere 3.8 cm. anterior to the left choroid glomus; this was thought to be calcification in the displaced glomus of the right

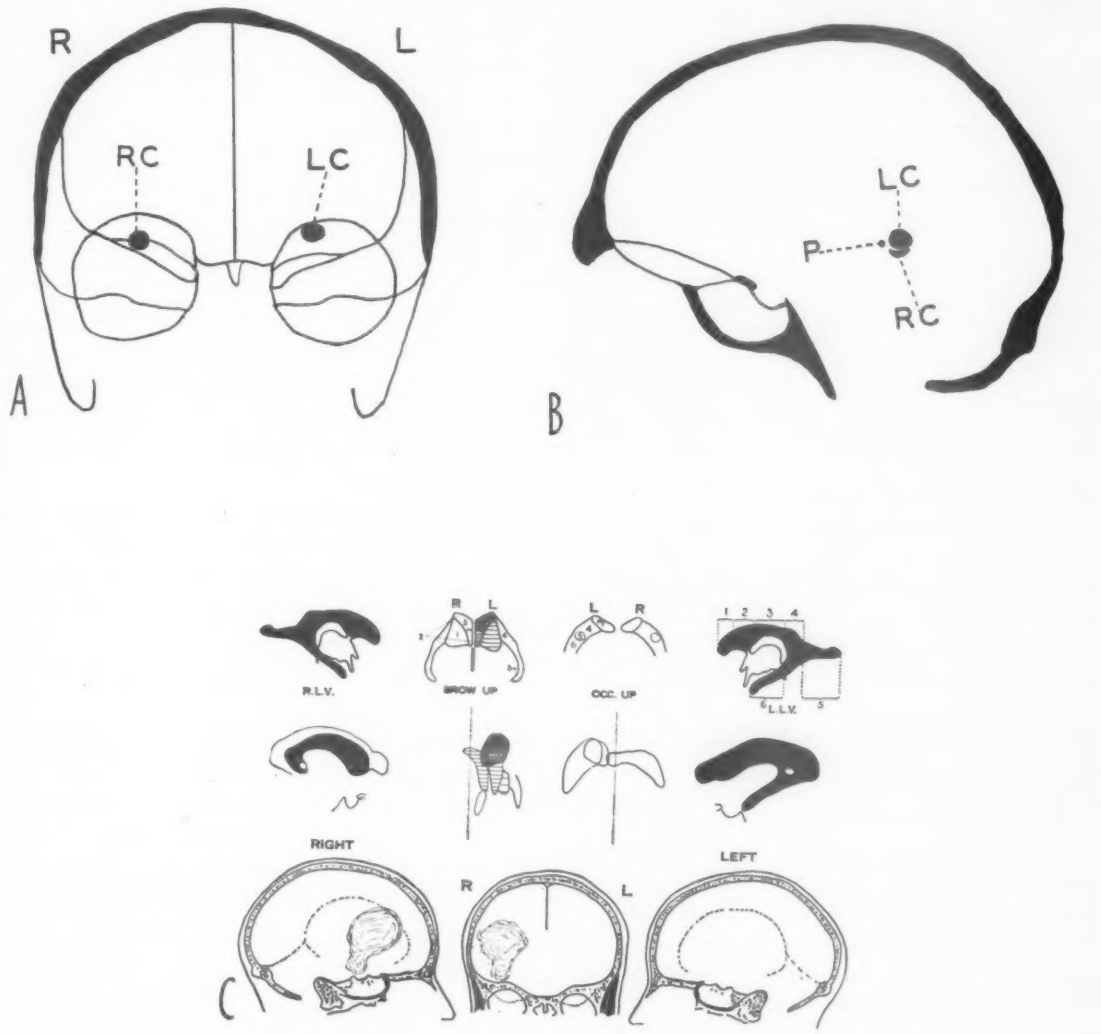

Fig. 9, *A*, *B* and *C*. Case IX. Very slight downward displacement of the right choroid glomus insufficient to warrant any conclusions. The lack of appreciable displacement is due to the anterior situation of the tumor.

The findings on examination were left homonymous hemianopsia, blurring of the upper halves of both optic discs, weakness of the left arm with impairment of sense of position of index finger and great toe on left side, and questionable left Babinski. Plain roentgenograms of the skull showed atrophy of the posterior clinoids and dorsum sellae. The pineal was calcified

choroid plexus (Fig. 8, *A* and *B*). From the flat films, a large right-sided expanding intracranial lesion situated posteriorly in the skull, possibly in the occipital region, was diagnosed. Ventriculography the next morning showed the septum pellucidum and the third ventricle to be displaced 1.5 cm. to the left of the midline. There was moderate dilatation of the left lateral

ventricle. Portion 3 of the right lateral ventricle ran obliquely from right to left. It was displaced forward, depressed, and somewhat compressed. Portion 4 was compressed, displaced at least 3 cm. farther forward than normal and spread out from side to side. There was some buckling of portion 6 and it was pushed forward. Portion 5 was depressed, compressed, and appeared elongated although actually its posterior tip was situated anterior to the corresponding region on the left side. It was also displaced laterally (Fig. 8, *C*). These findings suggested an expanding lesion situated on the right side of the cranium adjacent to the posterior part of the falx. At operation later the same day a meningeal fibroblastoma was removed. This was adherent to the falx and the median longitudinal sinus on the right side, and extended down to rest on the tentorium of the cerebellum. It was of large size and seemingly completely encapsulated. There was also a tiny tumor nodule attached to the dura at least 2 cm. from the main tumor mass. This latter tumor measured about 1 cm. in diameter.

CASE IX. I. E., male, aged fifty-five, was admitted September 12, 1939. There had been generalized headaches, more in the front of the head, with pins-and-needles sensation and attacks of dizziness with falling—usually to the right—for the previous six weeks. Associated tinnitus had been present for the same length of time. Physical examination revealed marked bilateral papilledema, right-sided plantar extension and reduction of the vestibular reaction.

Plain roentgenograms of the skull made on the day following admission revealed definite absorption of the posterior clinoids and dorsum sellae as well as beginning absorption of the posterior portion of the floor of the sella. Very faint pineal calcification was shown in the lateral films and this measured 1 cm. posterior to the normal zone as well as slightly below it. There was also bilateral calcification, apparently in the glomus of each choroid plexus. These latter calcified areas presented no real displacement although the right was very fractionally lower than the left (Fig. 9, *A* and *B*). Additional calcification, somewhat linear in nature, was shown deep in the right cerebral hemisphere, apparently in the inferior portion of the right frontal lobe. Subsequent ventriculography showed a shift of the septum pellucidum amounting to 1.8 cm. from right to left. The right lateral ventricle was compressed and portion 1 was displaced posteriorly over 1 cm. Portion 3 ran obliquely. There was hardly any displacement of portion 4 although it was smaller than the corresponding portion on the opposite side. It was not possible to visualize portion 6 (Fig. 9, *C*). Operation revealed an oligodendroglioma involving the right frontal and temporal lobes. The lack of displacement of the calcified glomus was evidently due to the anterior situation of the tumor.

COMMENT

In 6 of the 8 patients who presented displacement of a calcified choroid glomus the expanding lesions were rapidly growing (4 glioblastoma multiforme, 1 metastatic carcinoma and 1 hemorrhage). It is notoriously difficult either to lateralize or localize such lesions by means of plain films, pineal displacement usually being the only evidence of their presence. In the hemorrhage case there were bilateral fractures, the depression being on the side opposite to the collection of blood. Only 3 cases showed any absorption of the sella turcica. In 2 instances the pineal body was not calcified and in a third it fell within the normal zones. There was no deformity of the sella in these latter 3 cases and except for the displacement of the calcified choroid glomus it was not possible to diagnose a tumor. All the displacements of the choroid were very obvious except in Case V where the calcification was unilateral. In 3 of the 8 cases marked choroid shift was shown in anteroposterior roentgenograms.

Injection of a cyst or ventriculography has been carried out in 5 cases and these findings are included as confirmatory evidence. Removal or biopsy has been undertaken in every case so that the position and type of the lesions are proved.

In every instance the tumor was situated in the posterior two-thirds of the skull, the most anterior location being the posterior portion of the frontal lobe. Case IX is included to show that a tumor situated more anteriorly, even though it be large, may fail to displace the glomus appreciably.

DISCUSSION

When calcification occurs in the choroid plexus, it is almost always situated in the glomus and when bilateral it is symmetrically placed in each cerebral hemisphere in the vast majority of normal skulls. Theoretically calcification need not occur in exactly the same position in each choroid glomus, but in actual practice such asymmetry seems to be very infrequent, or at least of such minor degree that it causes no confusion. Macroscopic calcification in the choroid plexus elsewhere than in the glomus is quite rare. Consequently even a comparatively minor degree of apparent displacement of such a calcified area suggests a nearby neoplasm. When rather marked migration occurs, the evidence is much more definite, particularly when the calcification assumes a characteristic rounded or kidney-shaped form. It is much more difficult to be certain of slight displacement of the glomus when it is unilaterally calcified and in such cases one should be very cautious in drawing definite conclusions.

It must also be remembered that as the two glomi are as much as 5 to 6 cm. apart, minor degrees of rotation of the skull away from the true lateral will simulate displacement. When the roentgen tube is centered over the sella turcica, as is usually attempted in skull examinations, and a true lateral roentgenogram of this region is obtained it is normal for the glomus of the choroid plexus on the side farthest from the film to appear to be situated slightly posterior to and slightly above the glomus on the side closer to the film. If there should be some rotation of the skull so that the temporomandibular joint on the side away from the film is situated anterior to the temporomandibular joint on the side toward the film, the two choroid plexuses may lie exactly superimposed. If the reverse type of rotation is present, the choroid plexus farthest away from the film will appear to be situated even more posteriorly than it will be in a direct lateral. These simple facts are important and before anterior, posterior, or vertical displacement is considered, the presence or absence of rotation or high or low centering in the lateral films should be carefully noted. It is quite essential that stereoscopic films be made, so that the calcium may be localized. Since the routine adoption of anteroposterior or posteroanterior stereoroentgenograms, it has been found that a very much higher percentage of calcified choroid plexuses (and also calcified pineals) can be recognized in these planes than was formerly possible. Occasionally, as in Case VI, medial displacement may be more easily recognized than forward displacement.

There may be little or no displacement of the choroid glomus when a large expanding lesion is situated fairly well forward in the frontal or temporal lobes, due mainly to the stabilizing effect of the falx. While the falx is comparatively shallow anteriorly it forms a much thicker resistant curtain posteriorly and it tends to prevent migration of the posterior portions of the lateral ventricles, except when nearby pressure is applied.

It must also be remembered that slight displacements of the choroid plexus cannot always be recognized as readily as similar displacements of the pineal.

In every case in which displacement of a calcified glomus has been noted, the expanding lesion has been situated in either the parietal, occipital, posterior frontal, or posterior temporal regions. In such locations the lesion exerts more or less direct pressure on the posterior part of the ventricle with resulting displacement (shift). Lack of demonstrable displacement of the choroid glomus is not sufficient evidence to exclude an expanding lesion in the anterior part of one cerebral hemisphere.

CONCLUSIONS

(1) Calcification occurs sufficiently frequently in the glomus of the choroid plexus to constitute a valuable aid in the diagnosis of expanding intracranial lesions, particularly those situated posteriorly in the

skull. Eight verified cases are submitted to support this contention, in seven of which the correct conclusions were drawn from the plain roentgenograms.

(2) While it is possible that calcification may not always occur symmetrically in each glomus, this would seem to be a rare circumstance.

(3) When definite displacement of a calcified glomus is shown, an expanding lesion may be lateralized and fairly accurately localized.

(4) Lack of appreciable displacement of either choroid glomus, even though both are densely calcified and visible in two planes, is not sufficient evidence to exclude a tumor situated in the anterior part of the skull.

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CANCER OF THE STOMACH IN THE YOUNG*

By GORDON McNEER, M.D.
NEW YORK, NEW YORK

GASTRIC cancer is usually considered a disease of middle and late life. So firmly is this concept fixed in the minds of most of us that when a young adult presents himself with symptoms, which if encountered in an older individual would inevitably and rapidly lead to a correct diagnosis, one usually refuses to admit the possibility of a fact that is perfectly obvious. In this way much valuable time is lost and as a result all too often the patient is lost in addition.

If we discard, for the present, youth as the greatest stumbling block in establishing an early diagnosis, and face the facts purely on the basis of the historical and other data which the patient gives, one finds that the young subject inflicted with this disease presents a clinical picture closely parallel to that of his older brother. Because of its comparative rarity gastric cancer in the young is shrouded in mystery. Studies of a large series in any one institution are impossible because of its infrequency. As a result, a large number of erroneous facts have been handed down through the literature along with many misconstrued case reports. Instances of congenital pyloric hypertrophy were reported as infantile gastric cancer. Duodenal cancer, extrinsic tumors and other diseases were included. Hence, it was decided to make a study of the entire world literature on the subject, going back to the source in every available case. Facts are therefore presented which have been gleaned from such a study.

MATERIAL

Five hundred and one cases under thirty-one years of age were accepted for statistical analysis. These may not represent all instances, but the source was confirmed in each case and all repetitions were carefully weeded out. Nearly one hundred other reports were not accepted for various reasons, inadequate proof of diagnosis being the most outstanding. In addition, 5 cases of primary gastric cancer seen at the Memorial Hospital are presented. In order to emphasize certain points, comparisons are drawn with the material at the Memorial Hospital, consisting of 682 cases of primary gastric cancer in all age groups.

AGE INCIDENCE AND DISTRIBUTION

Finsterer mentioned 12 instances of juvenile gastric carcinoma among 535 operations for this disease. Osler and McCrae speak of 6 cases, or 4 per cent, of their series of 150. Smithies mentions 18 of a total of 917 case histories. Welch's figure of 2.8 per cent of 2,038 patients suffering with gastric cancer is certainly a fair estimate of the incidence of this disease in the young. Among the 682 cases of gastric cancer studied at the Memorial Hospital there were 5, or 0.7 per cent.

As would be expected, gastric cancer is extraordinarily rare in childhood, only 2 such cases being accepted in this series. After the age of fifteen, the incidence rapidly increases for each five-year period until twenty-six to thirty—with 272 case reports found suitable for study, as shown in Table 1. All the young patients seen at the Memorial Hospital were between twenty-four and thirty.

SEX

The sexes are equally involved: males, 46.7 per cent; females, 48.1 per cent, as opposed to a 3:1 ratio in adults. The sex was not given for 5.2 per cent of cases. Four females and one male were seen at this clinic.

HEREDITY

In 8 cases, either mother or father died of gastric cancer. In 3 one grandparent suf-

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fered from this disease and in 2 additional instances at least three members of the preceding generations were victims of carcinoma ventriculi. This represents 1.9 per cent of all the accepted cases.

RACE

Although Takenouti mentioned 54 instances in the Japanese literature, only 8 case reports were appended. Three reports in the Chinese literature were studied and one each in the colored and Mohammedan races.

SYMPTOMATOLOGY

Investigation of this phase of the study strongly suggests that the disease closely resembles its counterpart in older subjects, with several interesting exceptions. Three hundred and thirty-three reports gave sufficient historical data on which to base these conclusions. At some time vomiting was an outstanding factor in 64.3 per cent; pain in 62.2 per cent. With the cases seen at the Memorial Hospital, the order was reversed, with pain in 69.6 per cent and vomiting, 44.7 per cent. In 9.2 per cent of the young patients (19 cases) complaining of pain, it was highly suggestive of ulcer. Pain after meals occurred in 47.1 per cent of the Memorial Hospital cases. Hematemesis, which occurred in 37 instances, was the initial symptom in 10 histories, or 15.2 per cent of those patients complaining of vomiting. This was a higher percentage than for the Memorial Hospital cases. Pain was by far the most frequent initial symptom, occurring in 45.6 per cent of the cases. This was likewise true for the Memorial Hospital cases, with 34.8 per cent. The next frequent initial symptom was vomiting, 19.8 per cent, as compared with 8.3 per cent for the Memorial Hospital cases. The infrequency of weight loss as a complaint is an unusual finding, 21.6 per cent, for it is the most constant single symptom in cases of adult gastric cancer, occurring in 85.1 per cent of the older patients.

Table I

GASTRIC CANCER IN THE YOUNG
AGE AND SEX DISTRIBUTION

Age	Total		Male		Female		Sex Not Given	
	Number	Per Cent	Number	Per Cent	Number	Per Cent	Number	Per Cent
Total	501	100.0	234	100.0	241	100.0	26	100.0
5 yr. and under	1	0.2			1	0.4		
6 to 10 yr.	1	0.2	1	0.4				
11 to 15 yr.	17	3.4	10	4.3	7	2.9		
16 to 20 yr.	59	11.8	33	14.1	26	10.8		
21 to 25 yr.	136	27.1	70	29.9	64	26.6	2	7.7
26 to 30 yr.	272	54.3	120	51.3	143	59.3	9	34.6
Under 31 yr., exact age not given	15	3.0					15	57.7

Many authors have been impressed with the fulminating character of juvenile gastric cancer. That such is not the case, any more than with older adults, may be shown by the data given in Table II.

Three hundred and eleven case reports gave sufficient information for the study of the duration of the disease from onset to the hospital admission. The general average of all cases was 12.4 months, certainly long enough for any disease to become manifest; in the older patients it was 12.2 months.

It is very interesting that those falling into the resected group averaged 14.0 months before operation, compared with 15.9 months in the Memorial Hospital cases.

Physical Findings. In 164 reports an adequate physical examination was recorded. In 117, or 71.3 per cent, an upper abdominal mass was palpated, justifying exploration wherever possible. There was an abdominal mass present in 47.2 per cent of the Memorial Hospital cases. In only 9 was ascites present, 5.5 per cent, in contrast to 2.6 per cent for the older patients. Cachexia was described as being present in only 14, or 8.5 per cent. This has been a source of great astonishment to many. Whether the youthful subject has superior powers of resisting the tumor and its effects for a longer time than the older patients is a matter for conjecture only. Certain it is that he takes a longer time as a rule to show the constitutional effects of the disease than the older subject.

TABLE II

DURATION OF GASTRIC CANCER

Type of Case	Cancer in the Young Collected Cases	Memorial Hospital Cases 1916 to 1939
Total cases....	501	682
AVERAGE DURATION OF SYMPTOMS		
Type of case	Months	Months
Non-operative...	8.8	10.3
Operative.....	13.7	13.5
Laparotomy...	11.1	13.0
Palliative.....	15.9	13.2
Resection.....	14.0	15.9
AVERAGE DURATION FROM ADMISSION OR OPERATION		
Non-operative...	1.4	4.3
Operative.....	17.3	9.5
Laparotomy...	5.5	6.4
Palliative.....	6.5	8.7
Resection.....	29.0	22.2
AVERAGE DURATION OF THE DISEASE		
Non-operative...	8.8	14.1
Operative.....	23.6	22.2
Laparotomy...	13.6	19.3
Palliative.....	23.4	20.9
Resection.....	30.3	33.4

TABLE III

DIAGNOSIS OF GASTRIC CANCER

Diagnosis	Cancer in the Young Collected Cases		Memorial Hospital 1916 to 1939	
	Number	Per Cent	Number	Per Cent
Total.....	501	100.0	614	100.0
Antemortem and preoperative diagnosis:				
Correct.....	321	64.1	597	97.2
Borderline..	7	1.3	1	0.2
Incorrect...	91	18.2	16	2.6
Postmortem diagnosis only.....	82	16.4		

Excluding cases operated upon prior to admission to the Gastric Service of the Memorial Hospital.

Laboratory studies were not carried out with the uniformity necessary for statistical analysis. Blood counts were reported in a rather haphazard manner and only a few stool examinations were described. Enough gastric analyses were reported to be of some service to this discussion. Of 86 analyses 38, or 44.2 per cent, revealed achlorhydria; and 29, 33.6 per cent, a normal amount of free hydrochloric acid. This compares very favorably with the findings in older adults except that there is normally a much higher incidence of achlorhydria in the latter, being as high as 30 per cent in persons over sixty who do not have gastric cancer.

Nowhere in this study were the results of examination given less credence than in the roentgenographic reports. On many occasions the roentgen reports were to the effect that the defects resembled cancer, but the subject was deemed too young to have the disease. In the face of obvious filling defects a diagnosis of benign gastric ulcer was made time and again.

The differential diagnosis often lay between benign ulcer and cancer, with the former usually given the preference. As a result many cases were treated by medication long after the curable stage had passed.

The roentgenological diagnosis was correct in 63.6 per cent in the young group, and 97.5 per cent in the Memorial Hospital cases.

A correct antemortem or preoperative diagnosis was obtained in 321 cases, or 64.1 per cent, whereas it was established in 97.2 per cent of the older patients (Table III). This is reasonably satisfactory when one considers the fact that in a high proportion of cases roentgenological methods had not been perfected. However, with the advantage of modern roentgenological technique, gastroscopy, and a higher index of suspicion, there should be a very distinct improvement in this figure.

TABLE IV

OPERABILITY OF GASTRIC CANCER

Operability	Cancer in the Young Collected Cases		Memorial Hospital Cases			
			Total		1916–1930	1931–1939
	Number	Per Cent	Number	Per Cent	Per Cent	Per Cent
Total	501	100.0	609	100.0	100.0	100.0
Non-operative	158	31.5	280	46.0	55.5	40.0
Operative	343	68.5	329	54.0	44.5	60.0
Laparotomy	112	22.4	96	15.8	10.2	19.3
Palliative	94	18.8	176	28.9	30.9	27.6
Resection	137	27.3	57	9.3	3.4	13.1

OPERABILITY

As a general rule, 50 per cent of the older group afflicted with gastric carcinoma were totally inoperable, whereas of the younger age group 31.5 per cent only were deemed hopeless. Of the rest, 22.4 per cent had laparotomy, 18.8 per cent palliative operations, and 27.3 per cent resections. This last figure compares very favorably with the older age group, and refutes the statements made by many that the disease is utterly hopeless, an opinion that we will show later is entirely erroneous. Only 13.1 per cent of the Memorial Hospital cases from 1931 to 1939 were resectable (Table IV).

The pylorus and distal segment of the stomach were involved in 219 of the 342 cases giving information, or 64.0 per cent. Hence from location alone a fair percentage lend themselves to curative surgical intervention. With the Memorial Hospital cases, only 37.5 per cent of the tumors were located in the distal segment.

In 219 cases specified types of pathological entities were mentioned. The more frequent were as follows: scirrhous, about 20 per cent; medullary, about 60 per cent, and ulcerocancer, about 15 per cent. For the Memorial Hospital cases: scirrhous amounted to only 1.5 per cent and ulcerocancer 0.7 per cent, while the medullary represented nearly 90 per cent. There were 5 instances of linitis plastica in the young, and 3 in the Memorial Hospital cases.

METASTASES

Of the 501 cases the presence of metastases was noted in 275. From this, one should not deduce the fact that the remaining 226 did not possess metastatic cancer at any time, yet it may well be a fact, as in about 30 per cent of the older age groups Stewart found no metastases at postmortem. The high incidence of Krukenberg tumors is rather interesting (50 instances, or 35.5 per cent of the females with evidence of metastases). At the Memorial Hospital there were only 5 cases of Krukenberg tumors, 4.4 per cent of the females. The perigastric nodes were involved in 25.0 per cent, the liver in 26.5 per cent, and the peritoneum in 22.9 per cent. In the Memorial Hospital

TABLE V

OPERATIVE MORTALITY OF GASTRIC CANCER

Type of Operation	Cancer in the Young Collected Cases			Memorial Hospital Cases 1931 to 1939		
	Total Cases	Operative Deaths		Total Cases	Operative Deaths	
		Number	Per Cent		Number	Per Cent
All operations.....	343	68	19.8	222	27	12.2
Laparotomy....	112	23	20.5	69	8	11.6
Palliative.....	94	23	24.5	104	11	10.6
Resection.....	137	22	16.1	49	8	16.3

cases a higher percentage involved the perigastric nodes (35.1 per cent), a slightly lower percentage the liver (24.6 per cent), and for the peritoneum only 11.1 per cent. A high incidence (28, or 10.2 per cent) of pulmonary metastases was observed, much higher than in older subjects; for example, 6, or 1.8 per cent, in the Memorial Hospital series. In many the pulmonary symptoms so masked the picture that the patient was thought to be suffering from tuberculosis until postmortem examination revealed otherwise.

Osseous metastases from gastric cancer are rare, yet in 12 instances they were definitely shown to exist. There were only 3 cases in the Memorial Hospital series.

COMPLICATIONS

In 13 instances the patient was pregnant and in 3 definite signs of gastric cancer developed in the puerperium. Three were aborted but the patient's disease continued to its inevitable conclusion. Two were subjected to caesarean section—one for twins. All 13 patients died of their disease very rapidly. There was only one case at the Memorial Hospital which developed in the puerperium, in addition to Case IV which is included in the series of case reports at the end of the discussion.

Thrombosis of an external superficial vein occurred in one instance.

In 2 cases there were two independent cancers in the stomach. In 4 the gastric cancer was complicated by a cancer of another organ: (1) carcinoma of the large bowel, (2) carcinoma of the breast, (3) carcinoma of the cervix, and (4) retinoglioma.

PROGNOSIS

The operative mortality was highest (24.5 per cent) in those patients subjected to palliative operations. In the resected group it was only 16.1 per cent (Table v). Here also the period of palliation averaged approximately twenty-four months (Table II). In other words, of the 115 survivors of resection, 20 per cent, or 23 patients, lived without evidence of disease over three years on the average. This is not as gloomy a prognosis as that encountered in the older age groups where only 15.9 per cent survived three years (Table VI).

Thirty-two patients, or 6.4 per cent of the total 501, survived operation for over three years. This represents 27.8 per cent of the resectables. Nineteen, or 3.8 per cent, lived five years (16.5 per cent of the resectables) and there were 16 long-term survivors. In the Memorial Hospital group of older patients, only 1.1 per cent of the total survived three years and 0.7 per cent lived five years.

CASE I. J. M., female, aged twenty-five, admitted on December 21, 1920. For two years she had complained of a gnawing sensation in the epigastrium which at first occurred several hours after eating but later immediately after meals. One year ago swelling of the abdomen was observed. In June, 1920, a bilateral salpingo-oöphorectomy was performed elsewhere. Microscopic examination of the excised specimens

revealed metastatic carcinoma of the Krukenberg type.

Upon examination in this institution indefinite masses were present in the hypogastrium and right para-umbilical region. A large, firm, rather fixed mass nearly filled the pelvis with infiltration extending down the anterior vaginal wall.

A gastrointestinal series revealed a gastric carcinoma of the scirrhous type.

One roentgen treatment to the pelvis was given on December 31, 1920. She did not return. Death occurred on March, 3, 1921.

Case II. L. M., female, aged twenty-five, admitted on February 8, 1922. Patient was perfectly well until eleven months ago when she began to vomit after meals. She underwent appendectomy in July, 1921, without benefit.

In December, 1921, a gastric resection was performed for a tumor extending from the pylorus to the fundus. Pathological examination revealed carcinoma of the linitis plastica type.

After operation she gained weight and generally felt improved. While at Memorial Hospital she was given roentgen therapy to the abdomen in February and April, 1922. Death occurred on July 30, 1922.

Case III. H. D., male, aged thirty, admitted on August 7, 1925. One month ago he first complained of crampy epigastric pains, nausea, vomiting, and an indefinite amount of weight loss.

Physical examination revealed nothing remarkable.

The blood count showed a definite secondary anemia (hemoglobin, 40 per cent; red blood cells, 1,800,000).

Gastrointestinal series demonstrated an extensive cancer of the lesser curvature with an hourglass constriction and retention at six hours.

Laparotomy on August 19, 1925, revealed numerous miliary nodules on the peritoneum and a large mass of omentum adherent to the stomach. Jejunostomy was performed. Pathological examination of tissue showed carcinoma Death occurred on August 28, 1925.

Case IV.* H. W., female, aged twenty-seven, admitted on December 3, 1928. In 1926 she first complained of epigastric heaviness. This was followed by anorexia, nausea, vomiting and constipation. She was delivered of a normal child, but her symptoms continued. Her physician felt a mass under the right rib margin which as a result of roentgen study was found to be caused by cancer of the pyloric antrum. On October 30, 1928 she was explored and a gastroenterostomy performed because the mass could not be removed.

Upon admission she was seen to be anemic and emaciated. In the epigastrium a large, hard, movable mass was palpated. Rectal examination was not remarkable.

Gastric analysis showed a complete lack of free hydrochloric acid.

The blood count revealed only a mild secondary anemia.

* This case has previously been reported by Fred W. Stewart in the *Bull. Memorial Hosp., N. Y.*, 1929, *1*, 13–17.

Table VI

GASTRIC CANCER

POSTOPERATIVE SURVIVAL FOR THREE YEARS OR MORE

Percentages Based on Resected Cases and Total Cases

Condition of Patient	Cancer in the Young Collected Cases				Memorial Hospital Cases 1916 to 1939			
	Cases	3 yr.	5 yr.	over 5 yr.	Cases	3 yr.	5 yr.	over 5 yr.
Survival for 3 years or more	32				7			
Resected cases:	115	27.8	16.5	13.9	44	15.9	9.1	9.1
With disease		7.8	2.6	2.6				
Without disease		20.0	13.9	11.3		15.9	9.1	9.1
Total cases:	501	6.4	3.8	3.2	609	1.1	0.7	0.7
With disease		1.8	0.6	0.6				
Without disease		4.6	3.2	2.6		1.1	0.7	0.7

Extensive gastric cancer was demonstrated by roentgen examination.

She was subsequently treated by the 4 gram radium element pack, employing a daily fractionated technique. There was practically no improvement as a result of treatment. Death occurred on April 9, 1929.

Postmortem, April, 10, 1929: *Gross*—Cancer of the stomach with metastases to aortic abdominal lymph nodes, transverse mesocolon, omentum and pelvic peritoneum. *Microscopic*—Gelatinous adenocarcinoma Grade 2.

Case v. M. DeS., female, aged twenty-four, admitted July 2, 1934. Six months ago she first complained of a gradual loss of weight. Anorexia and vomiting soon set in. Gastroenterostomy was performed elsewhere five weeks ago, but vomiting continued.

Physical examination revealed an extremely emaciated young woman. A healed right upper rectus incision was seen. There was generalized lower abdominal fullness and tenderness. She was obviously in extremis upon admission and died on July 16, 1934.

At postmortem a diffuse generalized peritonitis was seen. Carcinoma of the prepyloric region with widespread lymph node metastases was likewise observed. The gastroenterostomy stoma apparently functioned well.

Tissue examination confirmed the gross and clinical diagnosis of carcinoma.

Case vi. R. P., female, aged twenty-three, admitted February 3, 1939. She complained of weakness and weight loss. On January 3, 1938, a partial gastric resection for carcinoma was performed elsewhere. Convalescence was uneventful and she was well until November, 1938, when she complained of pain in the right lumbar region and right abdomen.

Upon examination February 3, 1939, there was a huge, immovable mass in the epigastrium beneath the scar. In addition metastatic cancer obviously filled the pouch of Douglas.

This case was not considered suitable for even palliative therapy at the Memorial Hospital and she was referred to her family physician for terminal care.

(This case is not included in the statistical analysis which was limited to primary gastric cancer.)

SUMMARY

A study of the literature on gastric cancer in the young yields the following points of interest:

1. The disease is comparable to its counterpart in older age groups in regard to duration, pathological entities, analysis of gastric contents, resectability and prognosis, contrary to nearly all statements on the subject.

2. A comparatively high incidence of occurrence of Krukenberg tumors and ulcerocancers exists.

3. Such a high resectability rate in a large series in which many cases found at postmortem alone were included suggests an even brighter outlook from radical surgical treatment if instituted early.

4. A relatively high incidence of pregnancy is noted and uniformly fatal outcome in its presence.

5. A high index of suspicion is necessary in the case of any youthful subject who presents symptoms presumptive of a diagnosis of gastric carcinoma in older patients.

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DISCUSSION

Dr. George T. Pack, New York City. The first two facts derived from this material are an appreciation of the relative frequency of gastric cancer in young subjects and the unexpected good prognosis which attends gastrectomy for cancer in youth. The experience of any one gastroenterologist is so limited that one must rely on the analysis of a group of collected cases of gastric cancer in the young in order to study its behavior.

The relation of cancer in general to old age and youth has long been of interest to me. Swift said: "Every man desires to live long but no man would be old." One of the penalties for the attainment of old age is an apparent increased liability to the development of cancer. The assertion has been made that if any man lived long enough and were spared from other diseases, he would eventually die from cancer. However, I have observed that cancer is not a common occurrence in extreme old age; in fact, after the complete attainment of the senile state, cancer in general is progressively infrequent, with the important exceptions of squamous cell carcinomas of the skin, penis, lip and buccal mucosa which are more frequent in advanced years in ratio to the general population living at these ages. The incidence of these particular cancers of similar histological type increases each year until the normal span of human life is reached. These cancers are usually induced by chronic irritants, therefore the passage of time may permit this phenomenon of cancer induction to take place. The belief that senescence alone is a dominating factor in cancer occurrence is due to a misinterpretation of the facts, chiefly a failure to appreciate "age incidence" in respect to mortality in general and the normal percentages for age distribution.

Cancer is not a biologic retrogression because

it does not occur in all old people, as does such a lesion as arteriosclerosis. It is an accidental rather than a natural step in the involution of the body. It is at the beginning rather than at the terminal period of old age that the greatest cancer hazard is usually present. The early involutionary period for some organs coincides with the time of greatest cancer frequency in these particular organs. The specific involution of a particular organ which has fulfilled its function, such as the thymus, occurs at a relatively early age and in agreement with this we find thymomas predominantly in young adults. The prostate gland atrophies at a late period (the male climacteric) and of all the glandular organs it is the one involved at the oldest average age. The placenta has the shortest life of any human organ, yet at times it is subject to the development of chorion-epithelioma at a time period after fertilization which corresponds to its old age stage of degeneration. At birth the placenta is a senile organ with calcareous, fibrotic, atrophic and vascular changes characteristic of old age; its cancerous transformation is in keeping with its anatomic age. Degenerations of tissues and organs brought about by toxins, by endocrine imbalances as in thyroid and ovary, by infections as in lymphoid and myeloid tissues, by trauma as in fascial and osteoid tissues, by chronic irritations as in skin, cervix uteri, penis, lip and tongue may precede and indeed influence the development of cancer in these areas. But the onset of cancer in such instances does not have the same specific relationship to age as do the previously mentioned physiologic atrophies, which occur at definite periods in the life history of the individual. Age then affords a longer time for the effects of the agent to be produced, whether this agent be a toxin, germ, internal secretion or physical irritant. There are many specific examples which can serve as proof of these assertions. Bilharzia infestation usually occurs in childhood, frequently in infancy; the twenty or more ensuing years to young adulthood often suffices for the production of Bilharzia carcinoma of the urinary bladder in relatively young subjects.

In considering the malignant tumors of youth, particularly those of epithelial structure, two groups of tumor types present themselves for analysis:

(a) A group of malignant tumors which are essentially peculiar to infancy, childhood, and young adulthood; they are infrequent in older people. The five most common representatives of this group are Wilms' adenomyosarcoma of the kidney, ocular glioma, teratoma testis, endothelial myeloma and thymoma. All of these tumors are cellular neoplasms, endowed with great propensity and capacity for growth.

(b) Certain malignant tumors which appear at early ages, are unusual in this precocity, because they are more common in mature years or middle and later life. When compared with similar tumors in older persons, these cancers in youth have hitherto been considered as more rapidly growing, highly cellular, undifferentiated, anaplastic neoplasms of greater malignancy. The history of the disease is usually short, the course rapid and the mortality high. Cancer of the stomach in the young is in this latter group and it is a source of great surprise to learn that the outlook for these patients is equally as good as in the older age group.

A study of possible etiologic factors in this group of young subjects might be very important and might be more possible to analyze than in the older ages. In the tumors of lower animals, in which the factor of heredity definitely exists, there is the tendency for the tumors to occur at progressively earlier years, as the frequency increases in these strains. Therefore one would reason that with the group of cases of cancer of the stomach in persons under thirty years, it would be expected to find a higher incidence of hereditary trends than the figures of the present article indicate.

The high incidence of normal acidity or hyperacidity occurring in these young people with gastric cancer is important, not alone from the standpoint of diagnosis, because there is a natural tendency for achlorhydria to occur in older people. It is generally known that patients with normal or increased acidity of the stomach have a better chance of surviving the operation of gastrectomy for cancer and moreover enjoy a higher percentage of five-year survivals without recurrence than obtains in the group of patients with achlorhydria. Perhaps this fact alone may explain the surprisingly good results following the surgical treatment of gastric cancer in patients under thirty years of age.

THE DIFFERENTIAL DIAGNOSIS OF INTESTINAL POLYPOSIS*

By JOSEPH FELSEN, M.D.
NEW YORK, NEW YORK

THE term "intestinal polyposis" has been used rather loosely to include two distinct clinical and pathological entities, entirely unrelated, and often giving rise to certain false implications as to pathogenesis, diagnosis and therapy. We shall attempt to differentiate these two conditions, namely, adenomatosis coli and polyposis cystica intestini.

ETIOLOGY

The cause of adenomatosis coli is unknown. Ribbert[14] suggests their origin in embryonal rests. Versé, Ewing, Lockhart-Mummery, Dukes and others are inclined towards an inflammatory basis for the development of these multiple tumors supplemented by some peculiar familial "anlage." The consensus appears to be that the condition involves a definite hereditary factor of the mendelian type. This is clearly evident in all of our cases in which a familial history could be traced. Two or more members of the same family are usually involved, three generations being affected in one instance. It is also of interest to note that cancer supervenes in a similar manner, often being the cause of death in individuals of the same or different generations.

Polyposis cystica intestini involves no hereditary factor whatever. Evidence has been presented indicating that the disease is acquired by contact and represents a late stage of bacillary dysentery.[2] It has been traced in the same, previously healthy individual from the day of onset as the acute phase of bacillary dysentery down to the chronic phase of polyposis cystica intestini, approximately one year later. Follow-up studies of the Jersey City epidemic of atypical Flexner dysentery reveal the incidence to be 10.7 per cent.[5] The familial tendency in polyposis cystica is due entirely to contact infection during the early infective stage of bacillary dysentery. In follow-up studies of more than one thousand cases of acute and chronic bacillary dysentery not one instance of adenomatosis coli has been encountered. This observation suggests that if a chronic inflammatory process precedes the onset of adenomatosis coli, it is probably not due to bacillary dysentery.

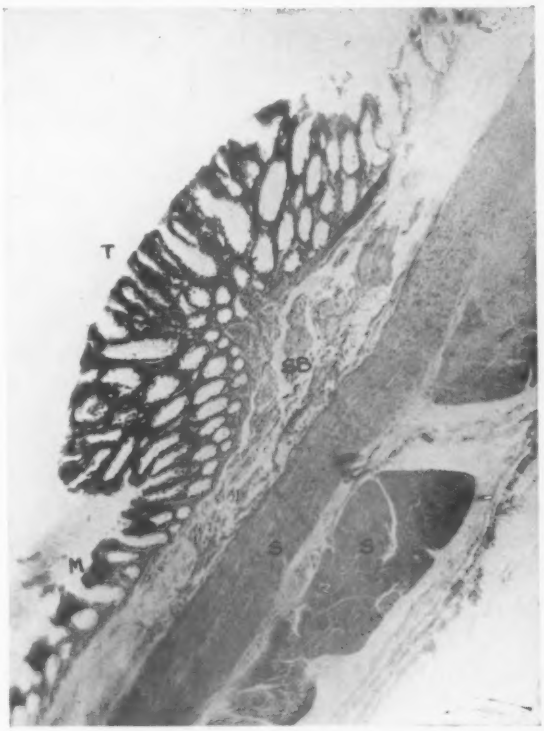

FIG. 1. Early stage of adenomatous polyp. *T*, tumor; *M*, normal mucosa; *SB*, submucosa; *S*, muscularis. Low power photomicrograph.

PATHOLOGY[1]

Adenomatosis coli is characterized by the development of multiple tumors which

* From the Department of Laboratories and Research, Bronx Hospital, New York. Read before the Section on Diagnostic Roentgenology, Third International Cancer Congress, Atlantic City, N. J., Sept. 12, 1939.

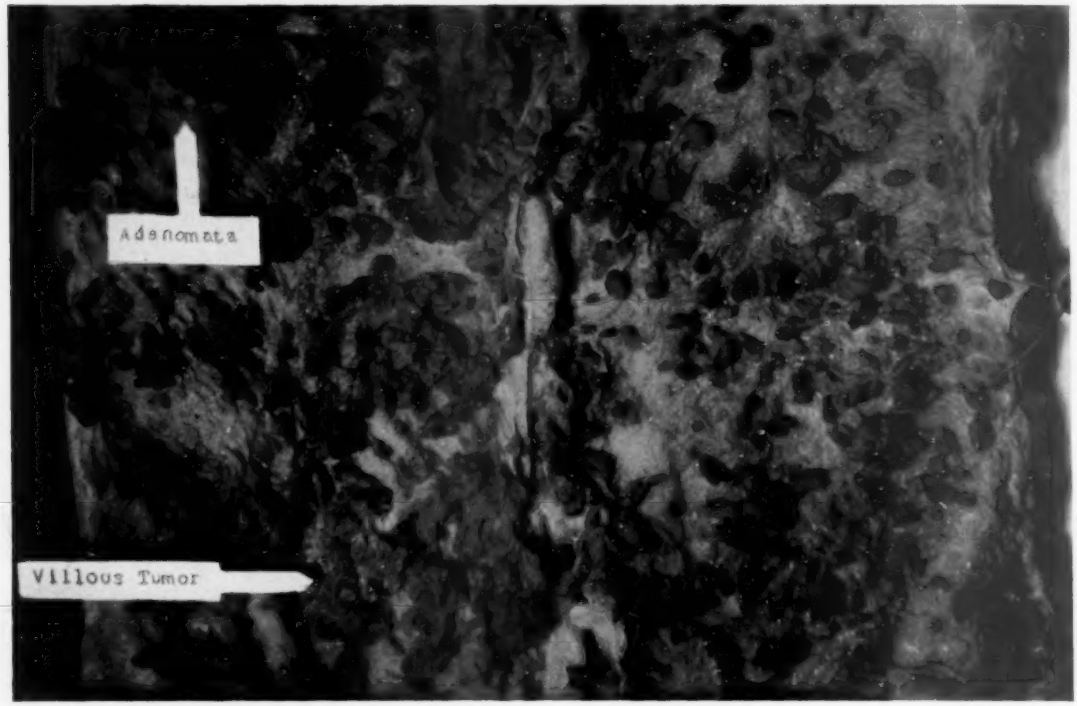

Fig. 2. No. 6443. Adenomatosis coli in young woman, aged twenty-one, whose mother, maternal uncle and grandmother had the same disease. Note associated villous tumor and areas of intact mucosa. Necropsy specimen.

arise from the mucosa of the colon. They are often so abundant that the mucosa intervening between individual tumors can only be discerned with difficulty. An important differential point, however, is that *normal* mucosa lies between the tumors. The adenomata start as localized proliferations of the mucosal epithelium with piling up of the glands. At first sessile and button-like in their gross appearance, they gradually extend on the mucosa and project into the lumen of the bowel until they appear as dark red, rounded tumors with delicate surface lobulations. As they increase in size the bowel attempts to extrude them as it would a foreign body. Since the mucosa and submucosa of the colon are rather loosely attached structures, the adenomatous tumor soon acquires a pedicle which often reaches surprising length, a tumor in the sigmoid sometimes appearing at the anus. In the pedunculated stage the adenomata have the characteristic appearance of being mushroomed on a pedicle of mucosa which has a rather narrow apex and broad base. Sometimes, possibly because of secondary intramural infection, a little dimple of intestinal wall, including serosa, is drawn into the base of the pedicle. In these cases snaring must be done with caution as otherwise an opening into the peritoneal cavity may result. Not infrequently the pedicle of an adenomatous polyp undergoes torsion with partial or complete necrosis of the tumor which is subsequently passed by rectum. The pedunculated tumors are generally benign. When malignancy supervenes, a previously benign adenoma with a short pedicle becomes sessile, spreads laterally, often undergoes central necrosis and eventually takes on the appearance of adenoma malignum or adenoma destruens. Both the benign and malignant adenomas possess a richly vascularized connective tissue stroma and bleed easily. Malignancy occurring on an adenomatosis basis remains rather well localized for a relatively long period, exhibits only a slowly invasive

tendency and, if resection is done at a reasonably early period, the tumor does not recur. Most of the surgical cures of carcinoma of the colon probably fall in this group. Multicentric malignancy is frequently observed in adenomatosis coli. Each nidus is separate and distinct, as revealed by serial sections of the intervening mucosa. The process is clearly not the result of spread from a single primary tumor along the submucosal lymphatic plexus with re-appearance as a secondary tumor on the mucosa at a distal point. We have never seen adenomatosis coli extend beyond the ileocecal valve, although involvement of the small bowel has been described by Lubarsch,[10] Niemack,[11] Ribbert,[13] Petrow[12] and Hauser.[9] Gatersleben[8] reports a case of multiple polyposis of the small intestine with malignant degeneration in a woman aged twenty-two. This occurrence must be extremely rare, particularly since adenomatous polyps are not seen with any appreciable frequency in the small bowel. Besides, the rarity of primary carcinoma in this location is well recognized. The earliest microscopic changes in the adenomatous polyp which point to malignancy consist in the multiplication of cell layers, atypical glandular morphology with nuclear hyperchromatism, imbalance of glands and stroma, loss of ducts and penetration of the muscularis mucosae. Even after penetration occurs, the submucosal lymphatic plexus acts as an effective barrier to the further spread of the disease for many months, perhaps for as long as a year. The prognosis in this type of tumor, relatively superficial and generally Grade 1, is therefore correspondingly good following resection. The entire subject of multiple adenomatous polyps of the colon requires further study. Adenomatosis coli has not been seen in the newborn, but individual tumors are often observed in young children. Single or multiple, they are often of the silent type. In our series of 955 necropsies, all ages, the incidence of two or more adenomatous polyps is 6.4 per cent.[7] Excluding the newborn, the incidence is 9.6 per cent. Whether adenomatous polyps are the precursors of *all* carcinomata of the colon as suggested by Schmieden and Westhues, Fitzgibbon, Rankin and others is perhaps still open to question. It appears certain, however, that adenomatous polyps do not invariably become malignant any more

Fig. 3. No. 6443. Adenomatosis coli. Note closely packed adenomatous tumors. Same patient as in Figure 2. Operative specimen.

than all ulcers of the stomach do. In this connection it may be of interest to mention a case of adenomatosis coli with malignant degeneration of two polyps at the splenic flexure.[6] This area was resected, but many adenomata were left intact in adjacent portions of the bowel because further operative procedure was deemed inadvisable. After seven years there are no symptoms or signs

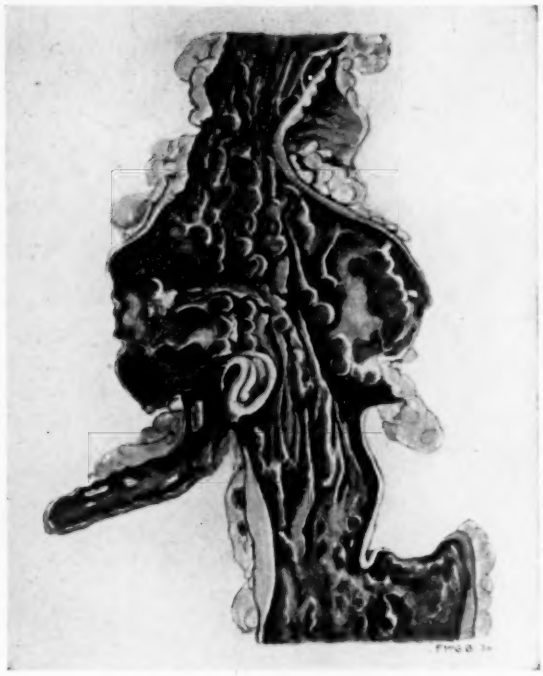

Fig. 4. No. 9273. Chronic ulcerative colitis with pseudopolyposis following bacillary dysentery. The entire colon, appendix and distal 30 cm. of ileum were involved. Water color shows proximal colon, distal ileum and appendix.

of malignancy.* The adenomata are undoubtedly still present as we have never seen these tumors resolve spontaneously. Adenomatosis coli appears to be associated with some underlying pathological "anlage" or tendency to the development of other intestinal pathology. It is axiomatic that "colon pathology generally is multiple." Villous tumors, submucosal lipomas, chyle cysts and diverticula are often associated lesions.

The gross pathological picture of polyposis cystica intestini is distinctive and without the slightest resemblance to adenomatosis coli. The condition is inflammatory, not neoplastic and represents one phase of the disease known as chronic ulcerative colitis which, in turn, is but a late manifestation of bacillary dysentery. The polyps are really pseudopolyps formed by swollen islands of intact epithelium which are surrounded by linear, geographic or serpiginous areas of ulcerated mucosa. The individual pseudopolyps are irregular in shape, soft and do not possess true pedicles. The surface is smooth, being entirely free of the delicate lobulations which characterize the adenomatous polyp. There is always an associated intramural infection, often with focal abscess formation. Besides ulcerated mucosa, the solitary lymph nodes and part of the inner circular muscularis are frequently destroyed. Healing may occur with extensive intramural fibrosis and epithelialization takes place from the mucosal islands of pseudopolyposis. In advanced cases, the bowel becomes a fibrosed, inelastic, narrowed tube of vascularized connective tissue. There is a characteristic sanguinopurulent discharge from the intestinal wall which is diffusely infiltrated by large numbers of polymorphonuclear cells. Hemorrhage is a frequent accompaniment of this disease due to necrosis of vessels by the infectious process. The disease often extends *beyond the ileocecal valve* into the ileum and may involve either the small or large intestine or both in a characteristic segmental fashion. Our studies indicate that in acute bacillary dysentery the pathological lesions are produced during the process of excretion of the dysentery toxin from the blood stream into the lumen of the bowel. There results a characteristic three stage progression of pathology: (1) punctate follicular hyperplasia; (2) punctate follicular necrosis; (3) discrete and confluent ulceration.[3] When the disease advances into the chronic phase, secondary nonspecific invaders, chiefly the enterococcus, hemolytic and non-hemolytic *B. coli*, enter the intestinal wall through the ulcers originally produced by *B. dysenteriae*, thus giving rise to a chronic nonspecific type of infection. The sites of predilection are the distal ileum, proximal and distal colon. We have never seen malignancy directly attributable to or even remotely associated with this condition, although it has occasionally been described. When present, it occurs as an independent disease, entire-

* A recent follow-up, approximately eight years after operation, reveals a mass in the liver which is undoubtedly carcinoma.

ly unrelated to the pseudopolyposis of chronic ulcerative colitis. The latter disease, moreover, occurs chiefly in the lower age groups, i.e. those most frequently exposed or non-immune to bacillary dysentery. Multicentric malignancy has never been observed in polyposis cystica intestini.

ROENTGENOGRAPHIC DIAGNOSIS

The roentgenographic signs of adenomatosis coli consist solely in the demonstration of the multiple, oval or round shadows representing tumors projecting from the wall of the colon into its lumen. The haustrations are preserved, the wall is of normal thickness and there is no stenosis. If present, the latter is localized and due to the size of the tumor or desmoplasia associated with malignancy. The diagnosis of adenomatosis coli is made with greater ease and certainty by sigmoidoscopy than by the roentgen ray.

The roentgenographic signs of polyposis cystica intestini consist of loss of haustration, defects in mucosal pattern, luminal stenosis and mural thickening. The picture is often characteristically segmental.

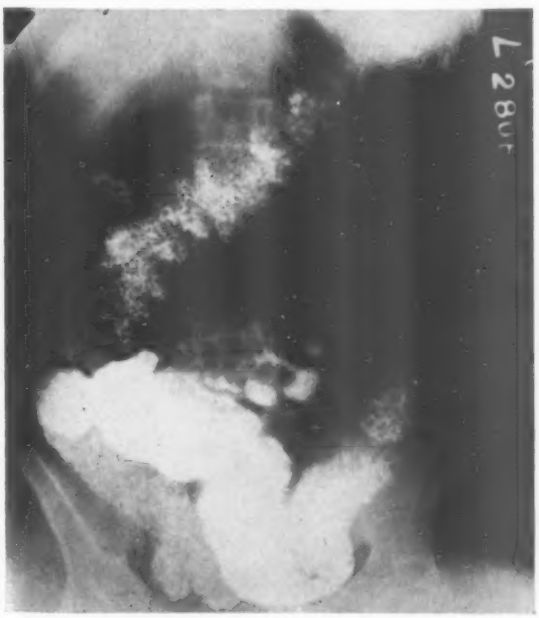

FIG. 5. No. 6443. Adenomatosis coli. Roentgenographic appearance of colon in same case as Figure 2. Note preservation of haustrations and absence of mural thickening or luminal stenosis.

FIG. 6. Roentgenogram showing loss of haustrations, stenosis, with pipe-stem appearance of colon and pseudopolyposis in case of chronic ulcerative colitis.

CLINICAL DIAGNOSIS

In adenomatosis coli there is usually a familial history of the disease. The chief complaints are bleeding from the bowel, some diarrhea or the protrusion of polyps during defecation. With malignant change, obstructive symptoms may occur. Loss of weight is usually not as prominent as a progressive secondary anemia from repeated hemorrhages.

In polyposis cystica intestini an epidemiologic history of acute bacillary dysentery may be obtained or *B. dysenteriae* actually cultured by the sigmoidoscopic crypt aspiration method.[4] Careful questioning will almost always elicit an initial acute phase of dysentery with sanguinopurulent stools, cramps and fever lasting ten days to two weeks followed by partial recovery. There then occur periodic exacerbations and remissions of the initial symptoms and signs.

These increase in severity and frequency until by the end of a year the patient is partly or wholly incapacitated. In severe cases, physical examination usually reveals an emaciated, anemic, sickly individual who exhibits a typical "mendicant's posture." In fatal cases, the patient is overwhelmed by toxemia, dehydration, acidosis and hypovitaminosis. Perforation with subsequent peritonitis occurs in a small percentage of cases and usually involves the sigmoid. In some cases, pinched off pseudopolyps are repeatedly passed by rectum.

SUMMARY

Adenomatosis coli is neoplastic, miliary and discrete adenomatous tumors studding the mucosa from the rectum to the ileocecal valve. There is a definite hereditary tendency of the mendelian type and the etiology is unknown. True multicentric malignancy is a frequent aftermath. There is no mucosal ulceration or mural fibrosis. Stenosis, if present, is local and due to the size of the tumor, malignant change or associated desmoplasia. There is no loss of haustration and the ideal therapy is complete colectomy.

Polyposis cystica intestini is an inflammatory lesion and consists essentially of small islands of intact mucosa separated by linear and geographic areas of ulceration. It is a pseudopolyposis and should more properly be called chronic ulcerative colitis. It is acquired and represents the late phase of initial acute bacillary dysentery. Mucosal ulceration, mural fibrosis and luminal stenosis often occur in a segmental fashion and may involve the small as well as large intestine. Loss of haustration is a characteristic roentgenographic sign and malignancy never supervenes. When found it is an independent and coexistent disease process. The ideal therapy of polyposis cystica intestini is the prevention of bacillary dysentery.

DIFFERENTIAL FEATURES OF INTESTINAL POLYPOSIS

Polyposis Cystica Intestini	*Adenomatosis Coli*
1. Acquired.	1. Hereditary factor of mendelian type.
2. The late result of bacillary dysentery.	2. Etiology unknown.
3. May involve any part of the small or large intestine, often in segmental fashion.	3. Generally limited to colon.
4. Inflammatory.	4. Neoplastic.
5. Does not become malignant.	5. Multicentric malignancy common.
6. Mucosa ulcerated.	6. Mucosa not ulcerated.
7. Lymph nodes destroyed.	7. Lymph nodes intact.
8. Mural fibrosis present.	8. No mural fibrosis.
9. Stenosis present.	9. No stenosis of lumen. If present, is local and due to size of tumor or desmoplasia associated with malignancy.
10. Loss of haustration.	10. Haustrations preserved.
11. Sigmoidoscopy reveals islands of mucosa delineated by ulcerated areas and sanguinopurulent exudate.	11. Sigmoidoscopy reveals multiple neoplastic adenomata with intervening normal mucosa.
12. Ideal therapy is the prevention of bacillary dysentery.	12. Ideal therapy is resection.

SIGMOIDOSCOPIC DIAGNOSIS

Sigmoidoscopy is the most valuable single method of differentiating adenomatosis coli from polyposis cystica intestini. The former reveals multiple solid adenomata, sessile or polypoid, with the characteristic appearance described above. One may be readily snared off for histopathologic diagnosis. In polyposis cystica, the rectosigmoid is usually involved and reveals a stenosed, rigid, ulcerated bowel with sanguinopurulent exudate covering both the ulcerated areas and the islands of pseudopolypoid mucosa which they delineate. The latter are sessile and irregular with smooth surface and rounded overhanging edges. They bleed freely on the slightest trauma.

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THE ROENTGEN DIAGNOSIS OF CYSTICERCUS

By A. I. DOMBROVSKY, M.D.
From the Department of Roentgenology, Higher Medical School
ROSTOV ON THE DON, U.S.S.R.

AT THE roentgen examination of a patient sent to our department with a suspected lesion of the lumbar spine, I happened to discover a very interesting accessory finding. Along both sides of the spine there were some rather dense soft tissue shadows with sharply outlined borders. Their length was about 9 mm., their breadth about 2 mm. The form of these shadows was spindle shaped; their longitudinal axis corresponded to that of the muscle fibers.

FIG. 1

At first I considered the possibility of some technical error, due to a defect in the film itself or to some stain on the intensifying screen. But on repeated roentgenograms these same shadows were found in the same position and presenting the same form as at the first examination. The number also remained the same (Fig. 1).

I suspected then that these shadows were calcified parasites located in the muscles of the back wall of the abdominal cavity. Systematic roentgen examinations have yielded the following findings:

All the extremities of our patient contain similar inclusions in the soft tissues. The greatest number are found in the femoral muscles (Fig. 2). The legs also contain a number (Fig. 3). The muscles of the arms and forearms show a smaller number of these shadows (Figs. 4 and 5). The hands as well as the feet are free from any visible inclusions. On the roentgenogram of the thorax one may see only single shadows here and there. As to the skull and the neck, one can easily find the shadows in the region of the posterior cervical muscles (Fig. 6). On the original roentgenogram, one of the shadows can be seen in the center of the lateral view of the skull under the sella turcica.

FIG. 2

For the confirmation of the diagnosis we undertook a biopsy and excised a small piece of tissue from the left breast where a rather large shadow was located among smaller ones (Figs. 7 and 8). In this tissue specimen we found a calcareous deposit with a diameter of about 3 mm. and of len-

Fig. 3

ticular form. The investigations carried out by Professor Krinitsky revealed that the exised tissue contained a partly calcified cysticercus.

A short history of the case follows.

M. D., working woman, aged forty-eight. The patient has never known her mother. She does not remember having been ill in her childhood. In 1920, typhus abdominalis and typhus exanthematicus; then influenza (Spanish type). No helminths. The patient is married and has three children. She has had three operations: (1) on

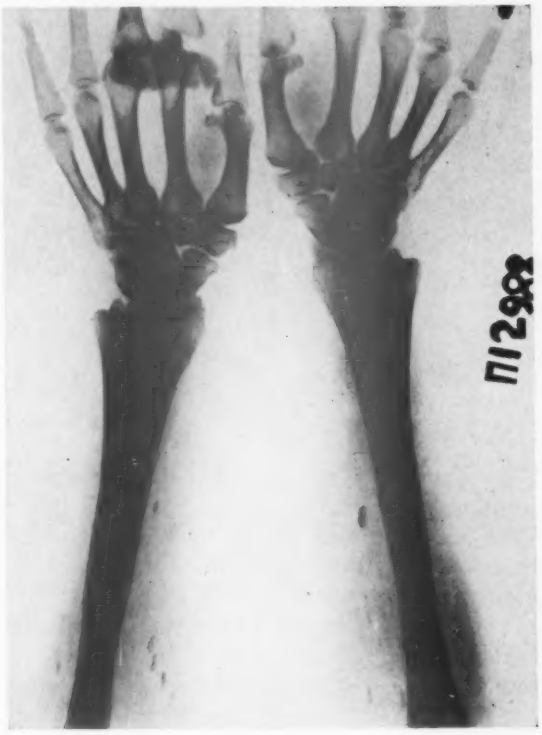

Fig. 4

the right knee because of accidental trauma; (2) on the toes of the right foot because of fractures of the bones, and (3) on the liver because of echinococcus. As to the last operation, which has the greatest interest, it has been impossible to gather any detailed information, it having been performed at another place. The patient herself told me (on the basis of what the nurses had related to her), that during this interference a "worm" had been extracted from her

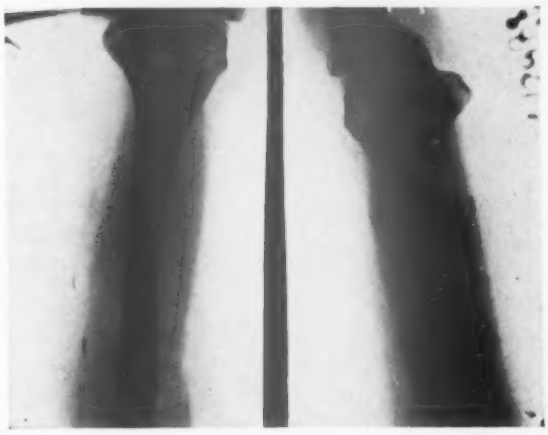

Fig. 5

FIG. 6

body instead of a "vesicle" as in the case of similar patients. After this operation, the patient had been sent to a health resort and had to take mud baths because of unsatisfactory healing of the wound. During the seventh bath the patient suddenly felt very weak and finally fainted. When she recovered her senses, she found herself in a hospital ward; the upper and the lower left limbs were paralyzed. Movement was gradually re-established, and two months after this accident the patient was strong enough to travel home alone from the health resort.

FIG. 7

During 1928 the patient had four or five similar attacks of loss of consciousness with convulsions, during which she bit her tongue and the mouth appeared contorted. But neither foam nor paralysis could be observed further. These attacks have never been noted since 1928.

In 1932 the patient contracted syphilis and underwent specific treatment. At the present

FIG. 8

time she complains of pains in the region of the heart and liver which become worse after meals.

The patient is a rather well-nourished woman of over middle height. Postoperative scars are seen on the skin in the region of the liver and of the right knee. The lungs are normal. The heart sounds are somewhat dull. The reaction of Romberg is positive; the rough muscular force is somewhat lower in the lower limbs. Tendon jerks in the lower limbs: right less than left; knee jerks, right greater than left, somewhat depressed. Abdominal reflexes, right greater than left. Achilles tendon reflexes, missing. Deep sensibility, normal; superficial sensibility, hypesthesia of the whole left side from the left median. Pupils, left greater than right; no re

action to light. Temperature, normal; pulse, 80. Blood picture: hemoglobin, 80 per cent; red blood cells, 4,000,000; leukocytes, 4,600; differential, eosinophiles 0.5 per cent, stab cells 6.5 per cent, segmented forms 55 per cent, lymphocytes 28.5 per cent, monocytes 9.5 per cent; blood Wassermann reaction one plus; spinal fluid Wassermann reaction negative. Urinalysis: straw colored, slightly cloudy, alkaline reaction; neither sugar nor albumin. In the sediment were found 11 to 15 leukocytes in the visual field; epithelial casts 7 to 9; crystals, triple phosphates in considerable numbers.

Neither helminths nor their eggs were found at the examination of the feces.

According to the literature, the first findings of calcified cysticercus bodies were made by pathologic-anatomists. Since the introduction into every-day practice of roentgen diagnosis there have appeared descriptions of such findings revealed by roentgenological investigations. In 1904 Stieda[1] briefly described such a case, and in 1913 Geipel.[2] In the last mentioned case a small number of bodies was present. Köhler[3] in his textbook gives the description of a similar observation. That of Schinz[4] mentions a case where single calcified cysticercus inclusions have been found in the muscles of a man aged fifty-nine. Finally, in 1929, Hinrichsen[5] published 2 cases in which a great number of calcified cysticercus inclusions had been found. The 6 mentioned cases are the only ones I have been able to find in the accessible foreign literature. As to the works published in the U.S.S.R. upon that subject, they do not contain, to my knowledge, any descriptions of this condition.

It is universally known that the cysticercus represents nothing but the larval stage of the band worm called *Taenia solium*. Man being the constant host, he is usually the bearer of the adult worm only, the latter living in the human intestine. The eggs of the band worm are excreted in the feces. If they happen to penetrate into the intestine of the intermediate host, which in the case of *Taenia solium* is the pig, round larvae develop out of these eggs which are called oncospheres and possess six hooks each. By means of these hooks, the oncosphere penetrates into the wall of the intestine where it settles down and is transformed later on into its following stage—the fin. In cases where these fins find their way into the small intestine of man (insufficiently cooked infected pork), they develop into the next stage, that of the band worm, *Taenia solium*. In rare cases (antiperistaltic movements, vomiting, self-infection because of untidiness or infection from outside), man can become an intermediate host for the band worm. The oncosphere having introduced itself into the human organism, it develops in the above-mentioned way into the stage of a fin in different organs and is then called cysticercus.

According to the frequency of their infection with this parasite, the human organs may be named in the following order: brain, eye, musculature, heart, abdominal cavity, spinal cord, breast.

Thus, infection with *Taenia solium* is possible in two ways: through food from other bearers or through self-infection. Some authors doubt the possibility of self-infection by means of antiperistaltic movements (Virchow, Hinrichsen), admitting only the oral method of infection.

The histories of Hinrichsen's cases do not contain mention of the patients having suffered from helminths in the past. Neither can such note be found in our own case.

The number of cysticercus inclusions in the human organism varies within very large limits from single ones to dozens and even hundreds. In our own case, we have been able to count over six hundred calcified cysticercus inclusions clearly seen in the films.

The single inclusion has the form of a vesicle, filled with fluid. Under the influence of the surrounding tissues, and chiefly also because on the death of the parasite, it takes the form of a spindle, sometimes it is oval. The longitudinal axis of this body corresponds very often to the direction of the muscle fibers. In our case it can be seen very clearly. In the muscles, the inclusions

are, for the most part, oblong in form, while in the breast, where the pressure of the surrounding tissues is not so strong, they have an almost round shape.

The dimensions of the calcified cysticercus vary greatly, even within the same organ. The above-mentioned authors have described cysticerci reaching the size of a lentil, a kernel of rice or even a bean. In our case the shadows have a length of from 2 to 9 mm., a breadth of 0.5 to 2 mm. The size of the cysticercus excised from the breast was 3 mm. in diameter.

It is usually thought that the fin lives about five or six years within the human body. When it dies, one observes either its resorption or its organization, followed by precipitation of calcium.

In the cases described previously, the authors had found cysticerci only in muscles or in subcutaneous tissue. They did not observe any clinical symptoms referable to the central nervous system; no mention could be found in the histories of their cases concerning the presence of cysticercus inclusions in the brain, the eye or in any visceral organs.

In our case we had positive information about lesions of the central nervous system in 1928 (palsies, attacks, etc.). At the present time a number are observed in the central nervous system, but these are apparently due to syphilis.

Even if cysticercus inclusions had existed in our patient in other organs besides the muscles, one must admit that at the present time they do not present any danger to their bearer, being already calcified. It should not be assumed that the parasites localized in the muscles have undergone calcification, while those distributed in other places have remained alive without giving any manifestations.

In our case the roentgenological differential diagnosis does not present any difficulties because of the great number of the inclusions and their characteristic shape. In cases of single formations, some doubts concerning the diagnosis may of course arise. In the first place, for instance, one might have considered phleboliths.

Trichinosis can be excluded, as the size of the shadows would have been very small, the maximum being but 1 mm., which is too small to be discerned roentgenologically. Separated muscles appear on the roentgenograms as if they had been sprinkled with sand.

In doubtful cases, one may have recourse to a biopsy, which, however, is not always easy since large and deep incisions are necessary. Hinrichsen, for example, had to make a 10 cm. incision before he penetrated to the fascia and extracted two calcified cysticerci. In our case we had no great difficulty in obtaining the desired inclusion as we had roentgenograms of the infected breast made in two projections.

While this lesion does not seem to present any danger to its bearer and therefore does not possess any great practical interest, its description may be justified by its rarity. The great number of cysticercus formations appears as a characteristic feature of our case, while the troubles observed in the past and referable to the central nervous system, and the "echinococcus" operation on the liver at the place occupied at the present time by the calcified cysticerci present considerable interest.

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PARTIAL HYPOPLASIA OF THE LOWER JAW (ACHONDROPLASIA PARTIALIS)

By KARL GOLDHAMER, M.D.
QUINCY, ILLINOIS

DISEASES of the temporomandibular articulation of noninflammatory type occur comparatively seldom in young individuals. Disregarding the sequelae resulting from traumatic or post-traumatic affections, it is chiefly the habitual luxation which renders roentgen examination of the temporomandibular joint indispensable. The more rare the illness is, however, the less is our experience regarding its diagnosis and treatment. In some rare cases all our knowledge often fails and we have to look for ways and means to find a necessary and adequate method of diagnosis and therapy. We sometimes make use of analogous diseases of the same organ or in patients of a greater age, or of the same affection situated in some other organ. The method of examination which gives the best information about the temporomandibular articulation is the roentgen examination, which, therefore, is of decisive importance for treatment.

The temporomandibular articulation, which is formed by the cavity of the temporal bone above, by the condyle of the lower jaw below, and by the articular disc situated between both of these, allows movement in all directions like a free joint. In the roentgenogram the condyle of the lower jaw occupies almost the whole fossa articularis in case the mouth of the patient is closed. On opening the mouth the condyle moves—as is known—forward in such a way that the upper end of the condyle is then situated closely behind the tip of the tuberculum articulare (articular eminence) of the temporal bone. In the roentgenogram the condyle partially covers the tuberculum articulare on account of the oblique projection; the condyle is projected also more in front, which may give the illusion of its moving in the direction of, or over, the tip of the tuberculum articulare. The spike which is seen at the boundary of the anterior surface of the condyle of the mandible to the fovea pterygoidea situated underneath corresponds to the place of insertion of the articular capsule and can be demonstrated almost regularly even in young individuals.

I wish to report one case, which seems to be extraordinarily rare (I could not find any similar case in the literature), which, however, is of great importance from a diagnostic, i.e. from a roentgen point of view due to its accompanying clinical symptoms.

The patient was a man, aged twenty, apparently quite healthy, an athlete, who stated that he had observed since his early childhood an unequal movement of both of his temporomandibular joints. Whereas on the left side he could not observe any abnormal phenomena he could often hear some slight crepitating noises on the right side. It was also possible to feel a fine friction on contact with the finger from the outside or putting the finger tip into the external auditory meatus. Disturbances did not occur until nearly six months ago, not even if the patient used the jaw violently. The patient's mother noticed that when he opened his mouth, and also when he closed it, the lower jaw was sometimes not exactly in the middle, but had moved a little to the left. A dislocation of the lower jaw on both sides or unilaterally had never been noticed. About six months ago the patient suddenly felt pain in the region of the right temporomandibular articulation. At the beginning the pain occurred only occasionally and extended towards the external auditory meatus. Later lasting spontaneous pain appeared, which, although not very violent at first, increased not only when chewing but also when speaking and in general with every movement of the lower jaw. The patient therefore consulted an otologist, who diagnosed the cause of the pain to be in the temporomandibular articulation and suggested a roentgen examination.

On palpating both articular regions of the jaw symmetrically one could observe a remarkable difference between the right and left side:

Fig. 1. View of closed teeth.

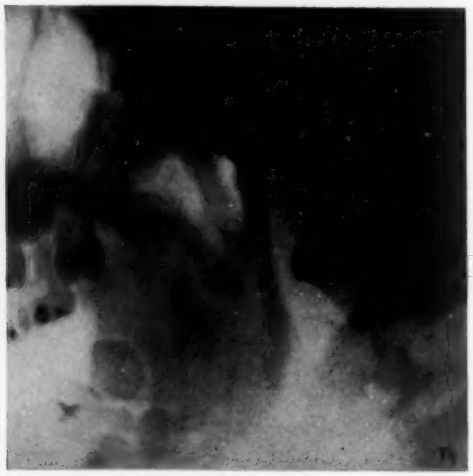
whereas at the left side could be felt a normal shifting of the condyle forward when opening the mouth and a normal lifting of the soft parts of that region, these symptoms were less pronounced on the right side. On observing the patient from the front we noted that when the mouth was closed the midline of the lower jaw frequently did not correspond with the midline of the upper jaw, but was somewhat displaced to the left (Fig. 1.).

The roentgen examination revealed at once the origin of the pain and the strange clinical findings. In the roentgenogram of the left side no deviation from the normal could be seen (Fig. 2). The left condyle shows a sagittal diameter of 10 mm. at the level of the insertion of the articular capsule; the height of the condyle of the mandibule (measured from the insertion of the articular capsule to the tip of the condyle) also amounts to 10 mm. Consequently the size of the area is about 100 mm.^{2} (These proportions correspond to the condyle observed in the roentgenogram, which are projected diagonally and do not correspond at all with the real anatomical measurements.)

On the right (affected) side (Fig. 3) the condyle of the lower jaw appears about 6 mm. deep and 4 mm. long; thus covering an area of about 24 mm.^{2} Consequently its area comprises about one-fourth to one-third of the area of the left condyle of the lower jaw. The right glenoidal cavity has—roentgenologically measured—a diameter of approximately 10 mm.; thus it is 4 mm. larger than the corresponding condyle.

Fig. 2. Roentgenogram of the left condyle of the mandible.

On opening the mouth the right condyle of the lower jaw advances to the normally developed articular eminence, but without surpassing it.



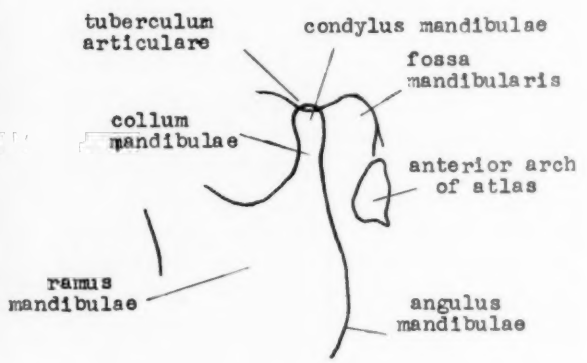


Fig. 3. Roentgenogram and tracing of the right condyle of the mandible.

In this position of the right mandibular condyle not only is its small size remarkable, but also its balanced, rounded form. The spike which corresponds to the insertion of the articular capsule is also missing.

Not only is the condyle of the lower jaw markedly smaller on the right side than on the left but the neck of the mandible is also much smaller and narrower. The mandibular angle is shallower and more rounded at the affected side. The distance between the tip of the condyle and the vertex of the angle amounts at the left (measured in the roentgenogram) to about 6.5 cm., at the right to only about 5.5 cm. A narrowing of the sagittal diameter of the ramus of the mandible or any reduction of the coronoid process of the mandible cannot be noted at the right side. The lower teeth are also quite normally developed on both sides. Thus it is only the dorsal part of the right ramus of the lower jaw which is less developed than normal.

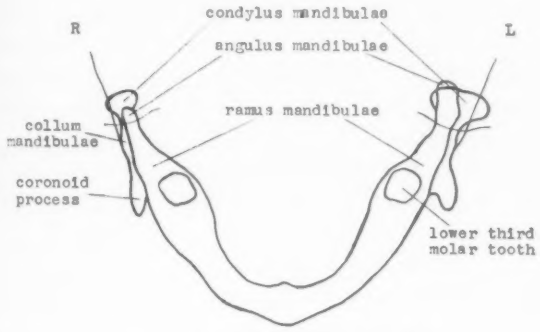


Fig. 4. Tracing of the lower jaw in axial projection.

In comparing views of both halves of the lower jaw, a marked difference is shown between right and left. In the axial projection (Fig. 4) the whole condyle of mandible appears considerably smaller on the right side. The horizontal diameter is about half of the normal diameter of the condyle on the left side. The inclination of the longitudinal axis of both condyles towards the median sagittal plane is, however, quite normal. The neck of the mandible is weaker and shorter on the right side. The position of the angle of the lower jaw in this projection is also remarkable. While its outline somewhat surmounts the condyle of the left lower jaw posteriorly, the analogous shadow-line only reaches the upper border of the neck at the right side. The ramus of the mandible is also not so thick on the right side as on the left.

The sagittal posteroanterior roentgenogram (Fig. 5) also shows a marked difference between the right and left of those parts of the jaw which are to be considered. Although the condyle and the neck of the mandible are smaller and narrower on the right side, they have a quite normal outline.

The measurements of this region on the living person—after the difference between right and

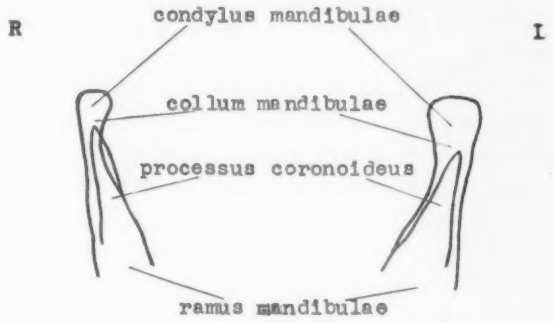


Fig. 5. Tracing of the lower jaw in anteroposterior projection.

left was noted in the roentgenogram—also yielded smaller figures of these parts. Even with the naked eye a slight asymmetry of the lower jaw appearing like a shortening of the right ramus mandibulae was seen.

Signs of traumatic, post-traumatic or inflammatory changes (which could be the cause of the deficient development of this part of the lower jaw) could not be distinguished in the roentgenogram. Neither did the history taken from the patient and from his mother yield any clue for this supposition.

Thus we have to deal with a case in which the dorsal part of the right ramus of the lower jaw is remarkably smaller than normally. The normal shape of these bones, their almost normal function and the absence of any signs of post-inflammatory, or possibly post-traumatic, changes are signs of deficient development, thus of congenital hypoplasia. The smallness of the glenoidal cavity may be a secondary sign. We are not, however, dealing here with a proportionate hypoplasia of the whole mandible (micrognathia) or of the whole joint, as the articular cavity and the articular eminence seem to be quite normally developed and do not show any remarkable flatness, but we are dealing with a change manifested only in the region of the condyle and ascending ramus of the lower jaw.

There is great difficulty in finding out which genetic period could be regarded as the origin of this malformation. We know that in some parts of the lower jaw during its development cartilage islands appear before the solid connective tissue ossifies. These parts are the tip of the condylar process, the angle and the posterior border of the bases of the lower jaw. It is remarkable that in our case the same parts of the ramus of the lower jaw are deficiently developed. This development seems to be at least partially based upon these cartilaginous islands. It might be possible that the development of these cartilaginous parts was hindered. The lower jaw (lacking these bone substances which normally are the secondary product of these cartilaginous islands) became smaller.

We can hardly give a satisfactory explanation as to the cause of this deformity. We know, however, a disease in which the disturbance of growth of the cartilage, beginning in fetal life results in lasting deformities of the skeletal parts which are developed from this cartilage; it is chondrodystrophia. We know several of its types: the mucous softening of the cartilage (chondrodystrophia malacica, chondromalacia), the disorderly proliferation of the cartilaginous cells which results in a protruding of the epiphyses (chondrodystrophia hypertrophica, chondrodysplasia, dyschondroplasia) and the deficient growth of the cartilaginous substance (chondrodystrophia hypoplastica, achondroplasia).

It may be that the last mentioned type of this disease of the cartilage does not affect the whole skeleton, but only certain parts. If this is the case, we could call it achondroplasia partialis or localisata, as only single parts of the cartilaginous system of a single organ are affected by their imperfect growth. The result is that a disturbance of growth is found in only one part of this organ.

Considering this fact, I tried to find some other cartilaginously pre-formed regions in our patient, which might manifest any imperfect development, such as the epiphyseal lines, other joints, and so on. I failed, however, to find a single place where imperfect development of the cartilage could be noted.

It is evident that this insufficiently developed temporomandibular articulation, during its period of growth as well as afterwards, cannot perform the complicated mechanism of chewing (as does a normally developed temporomandibular joint). This unilateral insufficiency of the temporomandibular articulation, which is the reason for the discrepancy between the efficiency of this joint and the strain put upon it, could best be compared with an arthritic change of the temporomandibular joint. As long as the lower jaw and the base of the skull are growing and developing (development of the teeth, especially of the wisdom teeth), the functional adaptation of the mandible might be adequate—the efficiency of the continuously developing lower jaw might be increased. When growth is concluded, however (our patient is now twenty years of age), the limit of the functional ability of adaptation and efficiency of the lower jaw is attained. That is the moment when the anatomical insufficiency manifests itself clinically and the disturbances of function and the symptoms begin.

It is evident from what has been described that the therapy can be only symptomatic. In our case we tried light massage, short wave treatments, but chiefly with careful roentgen therapy, to attain freedom from pain. The results, however, were only temporary. Now after two years of therapy, the condition of the patient remains unchanged.

SUMMARY

A report is given of a male, aged twenty, who revealed on roentgen examination a deficient development of the condyle and ascending ramus of the lower jaw. This condition may be called achondroplasia partialis. Clinically, the patient showed localized pain in the right temporomandibular articulation, upon which various forms of therapy had little effect.

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THE TREATMENT OF CARCINOMA OF THE BREAST BY INTERSTITIAL IRRADIATION*

By ROSCOE W. TEAHAN, M.D.
Jeanes Hospital
PHILADELPHIA, PENNSYLVANIA

IN 1920, Lee[26] at Memorial Hospital began to use radium emanation in interstitially placed platinum needles in the treatment of primary breast carcinoma. In the following year, Pfahler[34] used interstitially placed radium element needles in conjunction with roentgen treatment for the same purpose. Schmitz[41] likewise was an early user of this type of treatment for cancer of the breast. Except for perhaps a few instances, in none of these early attempts to treat cancer by interstitially placed radium was this the principal treatment. It was nearly always combined with some other form or was definitely used as a secondary treatment. Except in Lee's cases, the needles or tubes contained relatively large amounts of radium or radium emanation, and the container was constructed of such thickness and filtration value as to allow much beta radiation to reach the tissues and produce a caustic effect. This, together with the amount of radium in each needle, made it necessary to leave the radium in the tissues for but a few hours at the most. A short time later, Lee[24] seems to have abandoned in many cases the use of the more heavily filtered needles in favor of glass seeds, the so-called bare tubes. For larger tumors he used the more heavily filtered needles. Later the bare tubes were abandoned, and heavily filtered needles or gold seeds were used. However, in nearly all instances roentgen radiation was a very important part of the treatment.

In 1922, Keynes,[17] at St. Bartholomew's Hospital in London, began to treat recurrent breast cancer with interstitially placed radium element tubes. Here, too, relatively large amounts of radium were used, and the results were not particularly good. In consultation with Dr. N. S. Finzi, it was decided to place small quantities of radium in relatively long and heavily filtered needles in such a manner that the linear intensity would be low, and all the beta radiation would be prevented from reaching the tissues. Because of the small quantity of radium in each needle and the heavy filtration, it was necessary to use many needles for each patient and to leave them in the tissues for several days to produce the desired effect. More recurrent cases were treated with the new needles, and the results were so good that in August, 1924, treatment of inoperable primary carcinoma of the breast was undertaken. Such favorable response was obtained that by August, 1929, ninety patients with primary cancers of the breast had been treated. With experience, Keynes' technique gradually changed from that of implantation of the tumor-bearing area to a systematic implantation of most if not all of the breast, the axilla, the lower portion of the supraclavicular region, and the sternal ends of the upper three or four intercostal spaces. The needles were left in position for five, six, or seven days. In some cases a second treatment was given, while in others any residual tumor was later excised. Biopsies were performed in each of the first 50 cases, and all of the patients were carefully examined periodically for evidence of recurrence or metastasis.

In his most recent paper, Keynes[23] reports having treated a total of 325 primary carcinomas of the breast. His technique has continued to be much the same except that now, in some instances, he excises the primary cancer before implanting the radium needles. Operable as well as inoperable

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breast cancers are treated, and Keynes states that for operable cases he prefers this method to radical breast amputation.

The principles underlying Keynes' method of radium treatment are:

1. Homogeneous irradiation throughout the entire area where cancer cells may be present.
2. Sufficient filtration of the radium to divert all beta radiation. Only penetrating gamma radiation should reach the tissues.
3. Protraction of the irradiation so that the cancer cells will be exposed to it during division, when they are most vulnerable.

These principles are among those advocated by Regaud and his co-workers at the Radium Institute of Paris for the treatment of cancer in general. It is not evident whether in 1924 Keynes and Finzi were familiar with Regaud's views, but in any case Keynes was the first to apply them to the treatment of cancer of the breast.

Following Keynes' success, other English surgeons treated cancer of the breast in a similar manner. One of the first of these was Stanford Cade.[6] He followed Keynes' technique very closely as far as the implantation was concerned, but in addition he applied radium externally on a Columbia paste plaque to the lower part of the neck and the front and posterolateral aspect of the chest, three or four weeks after the interstitial needles had been removed. He refers to his treatment as a "radium Halsted," but the radiation actually extends over a much wider area than that covered by a radical breast amputation.

Fitzwilliams[11] modified Keynes' technique principally by placing the needles in the form of a grid beneath and about the primary cancer, and by using longer needles in the axilla. Some of the latter are placed directly behind the clavicle in such a manner as to extend from the axilla up into the supraclavicular region. He, too, makes use of an external radium applicator after removal of the interstitial needles.

Birkett's[4] principal modification of the method is exposure of the axillary contents by an incision for more accurate placing of the needles in this area.

Cheatle[8] first measures the breast and the cancer, and marks the size of each on a clay surface. He uses longer needles, some of which are 8.75 cm. in length. He estimates that each needle will destroy all of the cancer cells within a radius of 1 cm. Blocks representing each needle and the space its radiation will cover are so arranged on the clay surface and on each other as to cover the tumor and breast thoroughly. Needles are then placed in corresponding positions in the breast.

Ward,[48] working at the Radium Institute in London, had used gold radon seeds and interstitial radium element needles containing larger amounts of radium about primary breast cancers to supplement external radium applications. After having become familiar with Keynes' work, he began to use similar needles. He places these in the breast and in the anterior axillary wall, but relies on an external radium applicator to treat the remainder of the axilla, the supraclavicular region, and the parasternal region. In two to three months an external radium applicator is used over the entire affected side of the chest, including the axilla and supraclavicular area.

Meanwhile, on this continent, Lee began the use of gold radon seeds and roentgen radiation, followed by operation. Later, Pack[33] implanted not only the breast, but the lymphatic drainage areas as well, with long gold tubes containing radium emanation. Roentgen radiation was used in conjunction with the gold tubes. Adair[1,2] used implanted radon to supplement roentgen radiation and surgery, and more recently he has increased the amount of radiation reaching the axilla by placing a tandem containing radium emanation in the anterior axillary wall.

Papers by American authors on the Keynes technique began to appear in 1931 (Costolow,[9] Kaplan,[16] Cutler[10]). A paper by Cutler discussed the subject at some length and gave 2 case reports. Others followed (Soiland,[43] Meland[28]), and within a

short time a considerable number of patients treated had been reported upon (Meland,[30,31] Martin,[27] Soiland,[44] Taylor,[46] Pfahler,[35] McKittrick[28]). Modifications in technique have continued, the two most important being: (1) The substitution of radium emanation for radium element (Kaplan,[16] Cutler[10]); and (2) the use of roentgen radiation either before or after the implantation (Meland,[30,31] Soiland,[44] Taylor,[46] Pfahler[35]).

In a recent paper, Henry[14] of Canada reports having treated 130 patients. He implants the needles first, then after five or six weeks applies radium externally. He appears to use more radium at each implantation than does Keynes. A few reports from other countries are found in the literature, but apparently the technique has been used chiefly in England, the United States, and Canada.

Interstitial radium radiation for the treatment of breast carcinoma by the Keynes method was first used at Jeanes Hospital in 1933. Sixteen patients, several with advanced disease and some who had received considerable previous treatment elsewhere, were treated. In 1936, more patients were treated, but all of these were given a series of roentgen treatments first. In 1938, roentgen radiation was omitted.

The 68 cases reported are divided into five groups, as follows:

Group I—A lump in the breast, without palpable lymph nodes.

Group II—A lump, with palpable nodes in the axilla.

Group III—(a) A lump in the breast, with palpable nodes in the axilla and the supraclavicular area. (b) A lump adherent to the skin or the chest wall. (c) A lump, with distant metastasis.

Group IV—Postoperative recurrences.

Group V—Prophylactic irradiation.

PREPARATION OF THE PATIENT

Every patient who is to undergo treatment for cancer of the breast should have a thorough preliminary physical examination. The extent of the disease must be determined as accurately as possible. This applies not only to the breast, axilla, and supraclavicular region, but to the lungs and liver as well. In addition, roentgen examinations of the spine, pelvis, shoulder girdles, and skull should be made. A blood count before radium treatment is very important. During the course of the irradiation, especially if roentgen treatment has given previously, the development of a leukopenia should be watched for.

Radiation sickness has occurred very infrequently among the patients treated at this hospital. Adequate elimination and ample fluid intake are important in preventing its development.

TECHNIQUE

Two types of platinum needles have been employed. One type is 44 mm. long and contains 2 mg. of radium; the other is 60 mm. long and contains 3 mg. of radium. It is to be noted that the linear intensity of the active length is the same for the two types of needles. All the needles have a wall

SUMMARY OF CASES

	Group I	Group II	Group III	Group IV	Group V	Total
Living and well	10	11	11	0	1	33
Died of carcinoma	0	0	20	3	0	23
Died of intercurrent disease	1	1	4	0	0	6
Living with carcinoma	0	1	3	0	0	4
Killed in accident	0	0	0	1	0	1
Died, circumstances unknown	1	0	0	0	0	1
	12	13	38	4	1	68

SUMMARY OF CASES

Case	Age and Sex	Date Original Visit	Clinical Picture	Pathological Diagnosis	Previous Treatment Elsewhere	Roentgen Treatment	Radium Treatment	Surgery	Result	
					GROUP I					
1	41 F	8/22/32	Lump, 4.5 cm. in diameter, in upper hemisphere.	Carcinoma simplex	None	None	4/5/33 15,824 mg-hr.	Tumor excised at time of radium implantation.	Well 6/2/39.	Alive 81 months
2	60 F	6/15/33	Lump, 5×3.5×3.5 cm., in upper inner quadrant.	Carcinoma simplex	None	None	6/19/33 21,120 mg-hr.	Tumor excised at time of radium implantation.	Well 4/12/39.	Alive 70 months
3	61 F	6/15/33	Lump, 7×5×5 cm., in upper hemisphere.	Carcinoma simplex	None	None	7/17/33 24,240 mg-hr.	Tumor excised at time of radium implantation. 6/23/36—Exploration disclosed only fibrous tissue.	Well 4/14/39.	Alive 70 months
4	64 F	6/26/33	Lump, 5×5×3.5 cm., in upper outer quadrant.	Carcinoma simplex	Five roentgen treatments, May, 1933.	6/26/33—500 r to 3 ports. Treatment stopped following development of sharp erythema.	7/28/33 23,040 mg-hr.	Tumor excised at time of radium implantation.	Died 7/26/35, circumstances unknown. Last seen 9/13/33.	Alive 25 months
5	81 F	7/ 9/36	Lump, 3.8×2×1.5 cm., in upper inner quadrant.	Carcinoma simplex	None	None	7/13/36 12,312 mg-hr.	Tumor excised at time of radium implantation.	Well 3/13/39.	Alive 32 months
6	53 F	7/15/37	Lump, 4×5×4 cm., in upper hemisphere, involving nipple zone, with retraction of nipple.	7/16/37 Adenocarcinoma 11/7/38 Adenocarcinoma	None	7/21/37—1920 r to 3 ports.	9/23/37 10,962 mg-hr.	11/7/38—Radical mastectomy for persisting tumor.	Well 5/26/39.	Alive 22 months
7	65 F	10/ 8/37	Lump, 10×8×5 cm., in nipple zone, with retraction of nipple and watery discharge.	1/18/38 Carcinoma simplex	None	10/12/37—1120 r to 3 ports. Course interrupted by development of abscess incised 10/29/37. 12/20/37—1760 r to 3 ports.	3/11/38 24,351 mg-hr.	Tumor excised 1/18/38.	Well 6/14/39, except for some atrophy of muscles of hand and pain. Patient is a diabetic.	Alive 20 months

8	57 F	3/ 1/38	Lump, 3×2.5×2 cm., in upper outer quadrant.	Carcinoma simplex	None	None	3/20/38 31,941 mg-hr.	Tumor excised at time of radium implantation.	Well 4/14/39.	Alive 13 months
9	62 F	5/23/38	Lump, 6×4×4 cm., in upper outer quadrant. Questionable attachment to fascia.	Adeno-carcinoma	Breast partially removed 12 years earlier. No histological examination made.	None	7/ 5/38 21,903 mg-hr.	Tumor excised at time of radium implantation.	Died 6/19/39. No evidence of cancer.	Alive 13 months
10	42 F	6/14/38	Lump, 3.5 cm. in diameter, in upper hemisphere.	Carcinoma simplex	None	None	6/21/38 26,051 mg-hr.	Tumor excised at time of radium implantation.	Well 6/26/39.	Alive 12 months
11	58 F	9/14/38	Lump, 5×4×4 cm., in upper outer quadrant and nipple zone.	Carcinoma simplex	None	None	10/ 3/38 26,450 mg-hr.	Tumor excised at time of radium implantation.	Well 5/5/39.	Alive 8 months
12	33	10/22/38	Lump, 1 cm. in diameter, near periphery of upper hemisphere.	Carcinoma simplex	None	None	10/25/38 19,227 mg-hr.	Tumor excised at time of radium implantation.	Well 5/19/39.	Alive 7 months
					GROUP II					
13	67 F	10/ 8/34	Visible lump, 5×6×3.5 cm., in upper inner quadrant. Mid-axillary lymph node 1 cm. in diameter.	Carcinoma simplex	None	10/8/34—2000 r to 3 ports. 5/24/35—2000 r to 3 ports.	1/15/35 13,775 mg-hr.	None	Died 6/17/35 of heart disease. No disease evident in breast or axilla.	Alive 8 months
14	61 F	7/25/36	Lump, 2 cm. in diameter, in upper outer quadrant. Base axillary gland 7 mm. in diameter, and mid-axillary gland 1 cm. in diameter.	Carcinoma simplex	None	8/5/36—2200 r to 4 ports.	10/22/36 15,048 mg-hr.	Tumor excised 7/28/36.	Well 5/15/39.	Alive 34 months
15	59 F	3/ 6/37	Lump, 5×5×4 cm., in upper hemisphere and nipple zone, with retraction of nipple. Base axillary node and mid-axillary node, each 1 cm. in diameter.	6/14/37 Carcinoma simplex. 11/22/37 No cancer found.	None	3/10/37—2080 r to 3 ports, 1920 r to one port.	6/14/37 14,448 mg-hr. 11/22/37 7,300 mg-hr.	11/22/37—Breast explored for persisting tumor.	Well 3/17/39.	Alive 24 months
16	51 F	7/28/37	Lump, 7 cm. in diameter, in upper outer quadrant, with involvement of nipple zone. Apical axillary gland 1 cm. in diameter.	Carcinoma simplex	Roentgen treatments—3800 r to each of 3 ports, September, 1935, to spring of 1936.	8/4/37—1400 r to 3 ports.	2/28/38 19,741 mg-hr.	None	Well 6/18/39. No evidence of disease in breast or axilla. 7/27/38—Chest roentgenogram disclosed shadow, now considered fibrosis in view of marked improvement in health.	Alive 24 months

GROUP II—*Continued*

Case	Age and Sex	Date Original Visit	Clinical Picture	Pathological Diagnosis	Previous Treatment Elsewhere	Roentgen Treatment	Radium Treatment	Surgery	Result	
17	70 F	9/10/37	Lump, 3 cm. in diameter, in upper inner quadrant, with retraction of nipple. Mid-axillary node 1 cm. in diameter.	Carcinoma simplex	None	9/10/37—1800 r to 3 ports.	12/ 6/37 18,866 mg-hr.	Tumor excised at time of radium implantation.	Well 6/21/39.	Alive 21 months
18	56 F	10/13/37	Lump, 2.5 cm. in diameter, in upper hemisphere. Mass, 2×3×3 cm., at base of axilla.	10/22/37 Carcinoma simplex 10/25/38 Doubtful remnants of carcinoma.	None	10/22/37—1760 r to 2 ports, 2290 r to one port.	2/11/38 19,012 mg-hr.	Tumor excised at time of radium implantation. 10/25/38—Radical mastectomy for axillary mass.	Well 6/14/39.	Alive 20 months
19	46 F	5/ 5/38	Lump, 5×4.5×4 cm., in upper inner quadrant. Base axillary node 5 mm. in diameter. Mid-axillary node 1.5 ×1.5×1 cm.	Carcinoma simplex	None	None	5/12/38 26,362 mg-hr.	Tumor excised at time of radium implantation.	Well 6/2/39.	Alive 13 months
20	51 F	6/29/38	Lump, 4×4×2.5 cm., in upper hemisphere and nipple zone. Mass of glands, 7×7×2.5 cm., at base of axilla. Mid-axillary gland 1 cm. in diameter.	Carcinoma simplex	None	None	7/ 6/38 22,264 mg-hr. 5/24/39 Radium pack 73,400 mg-hr.	Tumor and axillary mass excised at time of radium implantation.	3/4/39—Palpable gland at base of neck; another in opposite axilla. 5/31/39—Some response to recent radium application. Breast negative.	Alive 11 months
21	49 F	7/19/38	Lump, 10×8×7 cm., in upper hemisphere, with retraction of nipple. Base axillary node 2.5 cm. in diameter.	Carcinoma simplex	None	None	7/28/38 25,080 mg-hr.	Tumor excised at time of radium implantation.	Well 6/16/39.	Alive 11 months
22	52 F	7/19/38	Lump, 9×5.5×4 cm., in upper outer quadrant, with retraction of nipple. Several enlarged glands in axilla. Base axillary gland 1.2×1 ×1 cm.	Carcinoma simplex	None	None	8/ 9/38 23,723 mg-hr.	Tumor excised at time of radium implantation.	Well 6/5/39.	Alive 11 months
23	42 F	8/23/38	Lump, 3×2.5×2.5 cm., in upper outer quadrant. Base axillary gland 8 mm. in diameter. Mid-axillary gland 1 cm. in diameter.	Carcinoma simplex	None	None	8/29/38 26,471 mg-hr. 10/24/38 Six gold seeds, 1 mc. each, in axillary node.	Tumor excised at time of radium implantation.	Well 6/5/39.	Alive 10 months

24	37 F	9/20/38	Lump, 6×4×3.5 cm., in lower inner quadrant. Mid-axillary node 4×4×3 cm.	Carcinoma simplex	None	None	9/21/38 26,943 mg-hr.	Tumor excised at time of radium implantation.	Well 6/15/39.	Alive 9 months
25	61 F	1/12/39	Lump, 4.8 cm. in diameter, in upper hemisphere and nipple zone, with retraction of nipple and marked reddening of skin. Mid-axillary node 3.8 cm. in diameter.	Carcinoma simplex	None	None	1/17/39 Radium pack 59,075 mg-hr. 5/ 8/39 5,520 mg-hr.	Simple mastectomy at time of radium implantation.	Well 6/16/39.	Alive 5 months
					GROUP III					
26	47 F	1/23/33	Lump, 7×5×3 cm., in upper outer quadrant and nipple zone. Attachment to skin, nipple, and underlying chest wall.	Carcinoma simplex	Roentgen (?) treatments, December, 1932.	1/25/33—1900 r to one port, 1750 r to 2 ports.	5/ 1/33 18,480 mg-hr.	None	Died 9/18/33. Autopsy disclosed metastases in lungs, liver, and peritoneum. Local disease uncontrolled.	Alive 8 months
27	47 F	3/ 7/33	Lump, 10.5×10.5×4.5 cm., involving all quadrants, with ulceration in nipple area and attachment to chest wall. Mass at base of neck, right side, and skin metastases.	Carcinoma simplex	None	3/14/33—1850 r to 2 ports, 1250 r to one port.	6/23/33 22,032 mg-hr.	None	Died 2/11/34. Local disease uncontrolled. Intra-abdominal metastases.	Alive 11 months
28	64 F	3/27/33	Lump, 4 cm. in diameter, in upper inner quadrant, with retraction of nipple and attachment to chest wall. Mid-axillary mass 6×4×4 cm.	Carcinoma simplex	None	10/9/34—1360 r to one port.	3/31/33 17,290 mg-hr. 3/1/34 7,812 mg-hr. 6/14/34 6,579 mg-hr.	None	Died 7/7/35. 1/18/34—Recurrence in axilla. 6/4/34—Recurrence in breast. 10/9/34—Recurrence in breast. Pulmonary metastases at time of death.	Alive 27 months
29	65 F	4/11/33	Lump involving almost entire breast, with attachment to skin and nipple, impending ulceration, and some fixation to chest wall. Axillary mass, 6×4×4 cm.	Carcinoma simplex	Approximately 35 roentgen treatments, beginning 1/4/32.	None	4/19/33 20,476 mg-hr.	None	Died 7/21/34. Breast and axillary tumors smaller.	Alive 15 months
30	54 F	4/18/33	Bulky tumor involving all quadrants, with attachment to skin and nipple, and ulceration. Mid-axillary mass, 4×4×6 cm., adherent to chest wall.	Carcinoma simplex	Incision and drainage.	6/15/33—1750 r to 3 ports.	8/ 4/33 14,440 mg-hr.	Simple mastectomy at time of radium implantation.	Died 1/22/35. No recurrence in axilla or chest wall. Metastasis to humerus and acromion.	Alive 21 months

GROUP III—*Continued*

Case	Age and Sex	Date Original Visit	Clinical Picture	Pathological Diagnosis	Previous Treatment Elsewhere	Roentgen Treatment	Radium Treatment	Surgery	Result	
31	57 F	4/28/33	Lump, 7.5×5.5×5 cm., in upper inner quadrant, with attachment to skin and nipple, slight ulceration, and fixation to chest wall. Base axillary gland 4.5 cm. in diameter. Mid-axillary gland 2 cm. in diameter. Metastasis to skin.	Carcinoma simplex	None	None	5/ 3/33 16,800 mg-hr. 7/ 5/33 13,824 mg-hr.	None	Died 10/28/34. Disease in breast never completely eradicated.	Alive 18 months
32	56 F	5/ 2/33	Lump, 6.5×5.5×4 cm., in inner hemisphere, with attachment to skin, nipple, and underlying chest wall. Mid-axillary node 2.5 cm. in diameter.	Carcinoma simplex	None	None	6/13/33 22,032 mg-hr.	None	Died 11/18/33, with metastases to pleurae, liver, and peritoneum. At autopsy, cancer cells found in breast, none in axilla.	Alive 6 months
33	47 F	6/10/33	Lump, 5×4.5×3.5 cm., in upper outer quadrant, with ulceration of skin, attachment to nipple, fixation to chest wall, and extension into mid-axilla.	Carcinoma simplex	Four "courses" of roentgen treatment, from 3/30/31 to 3/13/33.	None	6/12/33 14,784 mg-hr.	None	Died 1/8/34. Tumor in breast had shrunken.	Alive 7 months
34	57 F	6/26/33	Lump, 4×2.5×2.5 cm., in inner hemisphere, with attachment to skin and retraction of nipple. Low mid-axillary and apical gland palpable.	Carcinoma simplex	Roentgen treatment 3/1/33 to 5/18/33	None	7/17/33 26,160 mg-hr.	None	Well 6/21/39.	Alive 72 months
35	45 F	12/ 6/33	Lump, 7×4.5×4.5 cm., in lower hemisphere, with slight attachment to skin and retraction of nipple. Base axillary gland 2 cm. in diameter.	Carcinoma simplex	Two "courses" of roentgen treatment, July and September, 1933.	9/22/34—1500 r to one port.	12/11/33 30,000 mg-hr.	None	Died 7/22/36. 3/13/34—Primary tumor had almost disappeared, but axillary metastasis larger. 3/23/36—Roentgenogram revealed metastasis to skull.	Alive 31 months
36	60 M	6/30/34	Visible lump, 5.5×4.5×4.5 cm., in upper hemisphere, with attachment to skin. Another visible lump, 6×5.5×5.5 cm., in mid-axilla with fixation to chest wall.	Carcinoma simplex	None	7/2/34—2000 r to 3 ports. 5/27/35—800 r to 7 ports for skin metastases.	8/31/34 12,864 mg-hr.	None	Died July, 1935. Generalized metastases. Small mass still present in breast, none in axilla.	Alive 12 months

37	62 F	6/17/35	Lump, 6×7.5×2 cm., in upper outer quadrant, with retraction of nipple and attachment to chest wall. Extension to the axillary vessels. Supraclavicular gland 2×1.5×1 cm.	Carcinoma simplex	None	None	7/22/35 16,738 mg-hr.	Simple mastectomy at time of radium implantation.	Died 5/9/36 with widespread metastases. No evidence of cancer in treated area.	Alive 11 months
38	53 F	12/10/35	Ulcerating mass replacing entire breast, with destruction of nipple and fixation to underlying chest wall. Three axillary nodes, each about 1.5 cm. in diameter. Palpable glands in opposite axilla.	Carcinoma simplex	None	None	12/23/35 27,864 mg-hr.	None	Died 2/4/36 of bronchopneumonia. Tumor in breast and axillary metastasis had shrunken considerably prior to death.	Alive 2 months
39	41 F	1/10/36	Lump, 6×4.5×4.5 cm., in nipple zone, with attachment to skin and nipple. Base axillary node, 1.5×1.5×1 cm.	Carcinoma simplex	Four "courses" of roentgen treatment 9/8/33 to 8/29/35.	None	1/20/36 24,947 mg-hr.	None	Died 5/26/36 of bronchopneumonia. Breast tumor had shrunken to than half original size.	Alive 4 months
40	42 F	4/16/36	Lump, 4×3.5×2.5 cm., in outer hemisphere, with attachment to skin and underlying chest wall, and retraction of nipple. Axilla negative, but two enlarged glands at base of neck.	Carcinoma simplex	None	None	4/21/36 17,928 mg-hr.	Simple mastectomy at time of radium implantation.	Died 8/8/36. Incision broke down. No cancer found at autopsy.	Alive 4 months
41	64 F	4/20/36	Lump, 6 cm. in diameter, involving most of breast, especially upper hemisphere, with attachment to skin and retraction of nipple. Mid-axillary gland 1.2×1.2×1 cm. and apical gland 1.5 cm. in diameter.	Carcinoma simplex	None	4/25/36—2400 r to 3 ports.	6/18/36 8,680 mg-hr.	Simple mastectomy at time of radium implantation.	Died 6/25/37. No evidence of disease in treated area. Metastases in chest and left femur.	Alive 14 months
42	43 F	5/ 7/36	Lump, 10×7×3.5 cm., involving most of breast, with attachment to skin and retraction of nipple. Base axillary node 2 cm. in diameter, and questionable apical node.	5/11/36 Carcinoma simplex 3/25/38 Carcinoma simplex	None	6/3/36—2400 r to 3 ports.	10/20/36 13,436 mg-hr.	3/25/38—Simple mastectomy because of recurring nodules.	Died 3/18/39. Metastases to opposite breast and axilla, and to chest. Widespread bone metastases.	Alive 34 months
43	55 F	6/24/36	Lump, 5×4×2.5 cm., in upper outer quadrant, with attachment to skin and retraction of nipple.	Carcinoma simplex	None	7/2/36—2200 r to 3 ports.	9/15/36 15,371 mg-hr. 9/25/37 13,630 mg-hr.	None	Died 9/17/38, with metastases to 10th and 11th dorsal vertebrae. No cancer found elsewhere at autopsy.	Alive 27 months

GROUP III—*Continued*

Case	Age and Sex	Date Original Visit	Clinical Picture	Pathological Diagnosis	Previous Treatment Elsewhere	Roentgen Treatment	Radium Treatment	Surgery	Result	
44	60 F	8/ 3/36	Lump, 18×12×10 cm., with extensive skin attachment and retraction of nipple. Base axillary node 1.3 cm. in diameter.	Carcinoma simplex	None	8/4/36—2200 r to 4 ports.	10/ 5/36 17,136 mg-hr.	None	Died 1/13/37 of pneumonia, following fracture of left femur. No cancer found at autopsy.	Alive 5 months
45	65 F	10/ 2/36	Lump, 14×8×6 cm., in outer hemisphere, with extensive attachment to skin and retraction of nipple. Base axillary gland 4×3×3 cm. Several small mid-axillary glands.	Carcinoma simplex	None	10/2/36—2200 r to 4 ports.	1/14/37 15,602 mg-hr.	None	Died 7/18/37. Primary tumor and axillary metastasis had each shrunken to half of original size. Four months before death numerous skin metastases appeared over skin of chest.	Alive 9 months
46	42 F	10/ 7/36	Visible lump, 2.5×2.5×1 cm., in upper outer quadrant, with attachment to skin and adjacent muscle. Base axillary gland 1.2×1×1 cm.	Carcinoma simplex	None	10/7/36—2000 r to 2 ports and 2600 r to one port.	2/ 3/37 8,880 mg-hr.	None	Alive 6/16/39, but metastases to skin of right chest and neck. Primary tumor and axillary metastasis have disappeared. Metastasis to 1st lumbar vertebra, pelvis, femora, and ribs.	Alive 32 months
47	57 F	2/ 5/37	Lump, 4×2.5×2.5 cm., in upper outer quadrant, with attachment to skin and retraction of nipple. Base axillary gland 3 cm. in diameter. Mid-axillary gland 1 cm. in diameter.	3/23/37 Carcinoma simplex 3/28/38 Radiation fibrosis	None	2/11/37—1920 r to 4 ports.	4/19/37 21,050 mg-hr.	3/28/38—Simple mastectomy for persisting tumor in breast and axilla.	Died 5/21/39 of heart disease.	Alive 27 months
48	56 F	4/ 4/37	Lump, 12×12×5 cm., in upper outer quadrant, with attachment to skin and nipple. Mid-axillary node 4 cm. in diameter.	Carcinoma simplex	None	None	4/ 7/37 2,088 mg-hr.	Simple mastectomy time of radium implantation.	5/26/38—Metastases to right ilium, ischium, and possibly 3rd lumbar vertebra. (Patient not heard from since this date.)	Alive 13 months
49	56 F	7/ 1/37	Lump, 3.5×3.5×1.5 cm., in upper outer quadrant, with attachment to skin and retraction of nipple. Lowest axillary node 0.8 cm. in diameter. Mid-axillary node 1 cm. in diameter.	Carcinoma simplex	None	None	7/ 8/37 Radium pack 41,200 mg-hr. 9/8/37 8,092 mg-hr.	None	Well 3/31/39.	Alive 20 months

50	49 M	9/27/37	Lump, 4×4×1.5 cm., in nipple zone, with attachment to skin. Base axillary node 1 cm. in diameter. Questionable mid-axillary gland.	10/1/37 Carcinoma simplex 6/14/38 Radiation fibrosis 9/15/38 Radiation fibrosis	None	9/27/37—2080 r to 2 ports and 3480 r to one port.	11/17/37 12,116 mg-hr. 1/12/38 7,571 mg-hr.	6/14/38 — Radical breast amputation for enlarging mass at base of axilla. 9/15/38— Injudicious use of arm opened axillary incision. Subsequent hemorrhage required ligation of axillary artery.	Well 6/20/39.	Alive 21 months
51	69 F	10/11/37	Lump, 12×10×6 cm., in nipple zone, with reddening of skin of most of breast, attachment to skin, and retraction of nipple. Lowest axillary node 1.5 cm. in diameter.	11/19/37 Carcinoma simplex Aug., 1938 Carcinoma simplex	None	10/11/37—2200 r to 4 ports. 5/9/38—800 r to one port.	1/3/38 28,333 mg-hr.	Mastectomy performed elsewhere, Aug., 1938.	Died October, 1938. 5/4/38 — Skin metastases followed by recurrence of breast tumor.	Alive 12 months
52	48 F	11/11/37	Lump, 2.5 cm. in diameter, in lower hemisphere, with attachment to skin and chest wall. Two small axillary nodes.	11/29/37 Carcinoma simplex 12/2/38 Radiation fibrosis	None	11/22/37—1920 r to 3 ports and 2400 r to one port.	1/11/38 16,791 mg-hr.	12/2/38 — Simple mastectomy for persisting tumor.	Well 5/31/39.	Alive 18 months
53	64 F	11/29/37	Lump, 3.5×4.5×2 cm., in nipple zone, with attachment to skin and retraction of nipple. Base axillary mass 4.5×4×2.5 cm.	12/8/37 Carcinoma simplex 7/14/38 Carcinoma simplex	None	11/29/37—2400 r to 3 ports.	2/18/38 18,962 mg-hr.	7/14/38 — Radical mastectomy for persisting breast tumor. No cancer found in axilla.	Well 6/15/39. Small unhealed area at base of axilla due to radiation.	19 months
54	66 M	1/10/38	Lump, 5×6.5×4 cm., involving all quadrants, with ulceration and destruction of nipple. Mid-axillary node 2 cm. in diameter.	1/18/38 Carcinoma simplex 5/12/38 Carcinoma simplex	None	None	1/18/38 24,198 mg-hr.	5/12/38 — Radical breast amputation for persisting tumor in breast and axilla.	4/18/39—Sinus at operative site. No evidence of cancer.	Alive 15 months
55	61 F	1/24/38	Lump, 9×5×5 cm., in upper hemisphere, with ulceration of skin and retraction of nipple. Mid-axillary node 2 cm. in diameter.	1/27/38 Carcinoma simplex 6/7/38 Carcinoma simplex	None	None	1/27/38 32,329 mg-hr.	6/7/38 — Radical breast amputation.	Well 4/18/39.	Alive 15 months

GROUP III—*Continued*

Case	Age and Sex	Date Original Visit	Clinical Picture	Pathological Diagnosis	Previous Treatment Elsewhere	Roentgen Treatment	Radium Treatment	Surgery	Result	
56	45 F	2/16/38	Lump, 7.5×6.5×3.5 cm., in upper hemisphere, with ulceration, destruction of nipple, and attachment to chest wall. Base, mid-, and apical axillary glands involved, largest 1.5 cm. in diameter. Metastasis to 4th lumbar vertebra.	Carcinoma simplex	None	None	2/28/38 24,823 mg-hr.	None	5/12/39 — Breast and axilla free of palpable disease. Vertebral metastasis unchanged.	Alive 15 months
57	74 F	3/17/38	Visible lump, 5×4.6×2.8 cm., in upper outer quadrant, with attachment to skin and fixation to chest wall. Mid-axillary mass of glands, 5×4.3×2.8 cm. Node at base of neck.	Carcinoma simplex	None	None	3/29/38 26,246 mg-hr.	7/25/38 — Permission for excision of breast tumor refused by patient.	Well 6/16/39.	Alive 15 months
58	71 F	4/28/38	Lump, 5×3.8×3 cm., with attachment to skin. Base axillary gland 1 cm. in diameter.	5/3/38 Carcinoma simplex 9/16/38 Carcinoma simplex	None	None	5/3/38 29,478 mg-hr.	9/16/38 — Simple mastectomy for persisting tumor.	Died 4/5/39. Healed incision opened and later became necrotic. No cancer found at autopsy.	Alive 12 months
59	68 F	6/21/38	Lump, 4 cm. in diameter, in upper outer quadrant, with attachment to skin and retraction of nipple.	Carcinoma simplex	None	None	6/22/38 30,435 mg-hr.	Tumor excised at time of radium implantation.	Well 6/21/39.	Alive 12 months
60	81 F	6/20/38	Lump, 19×12×12 cm., in upper hemisphere and nipple zone, with ulcerated area 7 cm. in diameter. Several enlarged base axillary glands. Metastases to 3rd, 4th, and 5th dorsal vertebrae.	Carcinoma simplex	Two roentgen treatments, September, 1937.	None	7/18/38 22,678 mg-hr.	Simple mastectomy at time of radium implantation.	Died 8/14/38 of heart disease.	Alive 2 months
61	65 F	7/ 6/38	Lump, 4.5×4.5×2 cm., in upper outer quadrant, with attachment to skin and retraction of nipple. Base axillary node 2.5 cm. in diameter.	Carcinoma simplex	None	None	7/18/38 22,560 mg-hr.	Tumor excised at time of radium implantation.	Well 5/16/39.	Alive 10 months

62	30 F	12/19/38	Lump, 3×2×2 cm., in upper outer quadrant, with attachment to skin and retraction of nipple. Mid-axillary node 1 cm. in diameter.	Carcinoma simplex	Roentgen treatments, 9/15/38 — 2000 r to 4 ports.	None	10/27/38 23,896 mg-hr.	Tumor excised at time of radium implantation.	Well 6/14/39. Roentgenogram of chest shows questionable fibrosis of lung.	Alive 6 months
63	66 F	1/30/39	Lump, 5×5×3 cm., in lower hemisphere, with attachment to skin and retraction of nipple. Base axillary node 3×3×2 cm.	Carcinoma simplex	None	None	1/31/39 24,011 mg-hr.	Tumor and base axillary gland excised at time of radium implantation.	Died 2/14/39 of coronary occlusion.	Alive 2 weeks
					GROUP IV					
64	64 F	2/20/33	Multiple subcutaneous recurrences attached to skin, involving an area 8×4×1.5 cm., in 2nd and 3rd intercostal spaces to outer side of mastectomy scar. Base axillary gland 1 cm. in diameter.	Carcinoma simplex	Radical mastectomy, March, 1932.	Treatment given elsewhere. March, 1933—one "course" to 3 ports. Aug., 1934—Additional treatment because of appearance of enlarged gland at base of neck.	5/12/33 15,624 mg-hr.	None	Died 10/19/35. Autopsy revealed no recurrence in breast or axilla, but widespread metastases in pleurae, lungs, peritoneum, and subcutaneous tissue of neck.	Alive 32 months
65	62 F	1/ 4/37	Apical axillary gland 2 cm. in diameter.	Carcinoma simplex	Simple mastectomy, 11/24/36.	1/4/37—1600 r to 3 ports.	10/11/37 12,520 mg-hr. (Axilla, supra- and infraclavicular regions.)	None	Died 2/6/38. Metastases to pelvis and femora. Axillary node had almost disappeared.	Alive 13 months
66	77 F	3/ 9/37	Lump, 7×3.5×0.8 cm., in 2nd interspace, with attachment to skin and fixation to chest wall. Arm swollen. Chest roentgenogram showed fibrosis or metastasis in underlying lung.	Specimen unsatisfactory.	Radical breast amputation, 1917. Recurrence removed in 1935. Roentgen treatments in 1935, 1936, 1937.	None	3/10/37 848 mg-hr.	None	Died 10/16/38. Area of recurrence had disappeared. Probable metastases to lungs.	Alive 19 months
67	60 F	7/13/38	Nodule, 1.5 cm. in diameter, in skin and subcutaneous tissues of 5th intercostal space.	Carcinoma simplex	Radical mastectomy, June, 1936.	None	8/22/39 24,599 mg-hr.	Recurrent nodule excised 8/1/38.	9/21/38—Patient killed in accident.	Alive 2 months
					GROUP V					
68	56 F	7/ 7/38	Patient referred for prophylactic radiation, following removal of breast 6/25/38. The axilla was not dissected. No carcinoma was clinically demonstrable.	Carcinoma simplex	Simple mastectomy, 6/25/38.	None	8/2/38 23,083 mg-hr.	None	4/3/39—No evidence of cancer.	Alive 9 months

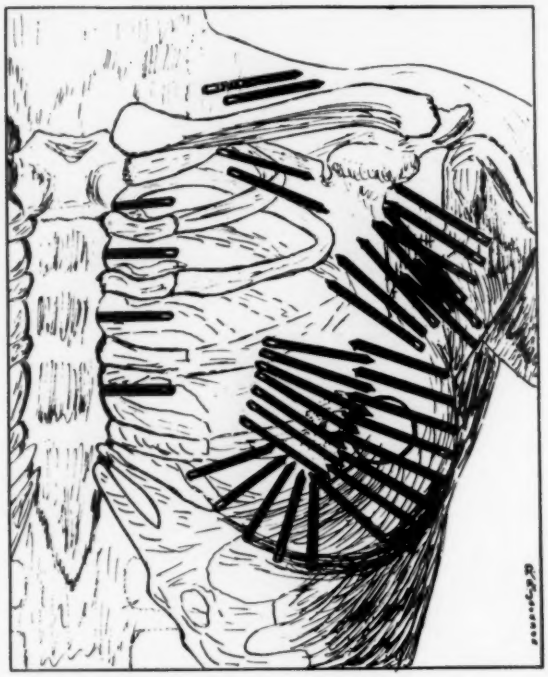

Fig. 1. Diagram showing approximate distribution of radium element needles. The circle indicates the position of the tumor.

thickness of 0.8 mm. It is desirable to have available for each patient about twelve of the 2 mg. and thirty of the 3 mg. needles.

The size and position of the tumor and the size of the breast must be taken into consideration in planning the implantation. The primary tumor and surrounding breast are first implanted. The longer needles are used for this, and they are placed in such a manner that they lie parallel to each other and 1.5 cm. apart on a plane just beneath the tumor. Two rows are generally used, the ends of the needles overlapping slightly. In a very thick breast, a row should also be placed superficial to the tumor. The area implanted is usually quadrilateral.

The implantation is carried upward and outward onto the anterior wall of the axilla, where four or five needles are placed just beneath the pectoral muscles in such a manner that the anterior wall is implanted and the needles converge slightly toward the apex of the axilla. Two or three additional needles are placed on each of the other axillary walls so as to converge at the apex. Injury to the axillary vessels will not result if the needles are inserted somewhat parallel to the vessels, without force and with the arm abducted.

Two of the 2 mg. needles are placed side by side just beneath the center of the clavicle and 1.5 cm. apart, their points directed toward the apex of the axilla. These are placed deeply and should lie close to the costocoracoid membrane.

Two 2 mg. needles are placed at the base of the neck above the clavicle and parallel to it. These are 1.5 cm. apart and should pass behind and to the inner side of the internal jugular vein.

A 2 mg. needle is placed in each of the four upper intercostal spaces in such a manner that the point of the needle lies against the posterior lateral edge of the sternum.

Finally, several 2 mg. needles should be placed in the lower part of the breast in the space between the implanted area and the epigastrium.

It seems desirable to avoid piercing the cancer during an implantation. It is to be remembered that the ends of the needles contain no radium, so that if carcinoma cells are transplanted by them, they may not receive as large an amount of radiation as might at first be expected. Moreover, forcing a radium needle into a hard tumor may bend or otherwise injure the needle.

For the implantation, nitrous oxide and oxygen anesthesia is administered, and the skin is prepared with iodine and Richardson's solution. The iodine should be removed carefully to avoid subsequent irritation of the skin. The needles are inserted through skin punctures, and the distance between the punctures is measured accurately. Before insertion, each needle is threaded with flexible monel metal wire, and these wires are tied together in groups before the dressing is applied. It is important to account for each needle at the end of the procedure. A diagram illustrating the size of each needle and its position in relation to the tumor, breast, nipple, clavicle, axilla, sternum, and ribs is very useful for purposes of reference.

At the completion of the implantation, a piece of the tumor should be excised for microscopic examination. Sterile dressings are then applied and the arm is supported at a right angle to the body until the needles are removed, at the end of eight to twelve days.

Many factors, such as the size of the breast, the size of the tumor, and the extent of involvement, must be considered in determining the amount of radium to be used and the length of time it is to remain in place. Among the cases reported here, the smallest number of needles used at any one time was six, the case being one in which only the axilla was implanted. The greatest number of needles used at one time was fifty-nine. The smallest quantity of radium used at one time was 12 mg., the greatest 134 mg. The needles were left in place for from 120 to 313 hours, the milligram-hours varying from 848 to 32,329. In 9 cases, more than one application of radium was made. In 3 of these, one of the applications consisted of external surface irradiation, in two before and in one after implantation.

REACTION

Surprisingly little discomfort is caused by the presence of the radium needles. During the period between implantation and removal of the needles, one change of dressing may be desirable, since this usually makes the patient more comfortable. Otherwise, the dressings should be disturbed as little as possible. In nearly all cases the needles can be removed without anesthesia, provided that a hypodermic injection of morphine sulphate, gr. $\frac{1}{4}$, has been administered forty-five minutes earlier.

Fig. 2. Case 3. Tumor in upper hemisphere of breast before treatment.

Fig. 3. Case 3. Tumor removed from breast with electrosurgical knife just prior to implantation of radium needles.

The first reaction consists of a reddening of the skin about each needle hole. This is evident in six or seven days. By the time the needles are removed, there is frequently a reddening over the entire irradiated area. This increases in intensity, and as a rule an epithelitis develops over the breast and in the axilla. The reaction reaches its height on about the eighteenth day, and healing is complete in four to six weeks. Within several weeks, light-brown pigmentation of the irradiated area is noted, and this may persist for a long time. Telangiectases are occasionally seen.

At first, the primary tumor softens somewhat and becomes more indistinct. Changes are usually evident within two weeks, and at the end of four or five months the tumor

Fig. 4. Case 3. Reaction to irradiation on twenty-fourth day following radium implantation.

will have disappeared or a small fibrous mass will remain. Metastatic glands are apt to be slower to respond than is the primary tumor. The epithelitis makes palpation very unsatisfactory, and when this has subsided thickening of the skin in this region likewise interferes with satisfactory examination.

POST-IRRADIATION COURSE

Frequent observation is advisable in order to note regression of the tumor, as well as to discover any recurrence. Edema of the dependent portion of the breast frequently develops following radium treatment. In the cases reported here, such edema has nearly always been due to the irradiation and not to a recurrence of the cancer.

A mass remaining in the breast four or five months after radiation treatment should arouse serious concern. Differentiation between residual carcinoma and fibrosis caused by irradiation is difficult. Keynes states that he is able to "distinguish confidently between fibrosis and recurrence." Cade,[7] on the other hand, contends that if a residual mass is present three months after the irradiation has been completed, it had better be removed. In a recent paper, McKittrick remarks about the difficulty he has in differentiating between recurrences and fibrotic or inflammatory masses in the breasts which have been treated. Among our patients, this differentiation has been most difficult. From our experience it is felt that in doubtful cases excision of the area or, probably better, simple amputation of the breast, is imperative. Simple mastectomy has been performed four times under such circumstances, and in 2 cases only irradiation fibrosis was present, but in each of the other 2 cases viable cancer cells were found. In 2 patients, induration in the irradiated area led to exploration five months and three years, respectively, after radium treatment. No cancer was found in either, and both are well. Another patient developed induration at the base of the axilla about one year following radium irradiation. Here a recurrence was mistaken for fibrosis, and when the error was discovered the disease was too widespread to permit excision. In 2 other cases, a mastectomy for a persistent lump in the breast was advised, but both patients refused operation. Eleven months have elapsed in each instance, with no apparent change in the size of the mass.

It is likewise difficult to determine the nature of masses in the axilla. Because of

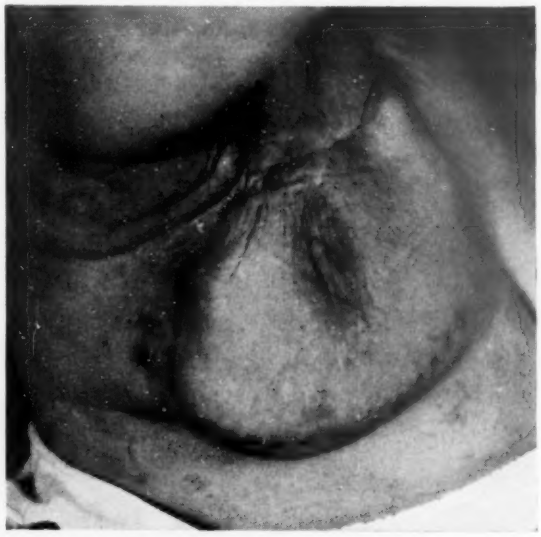

Fig. 5. Case 3. Appearance of breast three years after radium irradiation.

the presence of a mass in the axilla or close to it, a radical mastectomy has been performed or attempted in 7 cases. Examination of the breast and axillary contents has disclosed the presence of cancer in 5 of these cases. In one only irradiation fibrosis was found, and in the seventh doubtful remnants of cancer.

Fibrosis of the underlying lung occurred in 3 patients, all of whom had received roentgen irradiation before having had an interstitial radium application. In none of these cases has a second application of radium been made.

Operation following interstitial radium treatment is a dangerous procedure as far as axillary dissection is concerned. In the operations mentioned above, it was not possible in any case to perform what is generally considered a thorough axillary dissection. The irradiation causes a matting together of the axillary structures in such a way that they can be separated only by sharp dissection, and there is great danger of injury to the large blood vessels when such dissection is attempted. In one case it was practically impossible to separate the axillary structures at all. Pack[33] found a similar condition in patients treated by means of gold radon transfixion tubes.

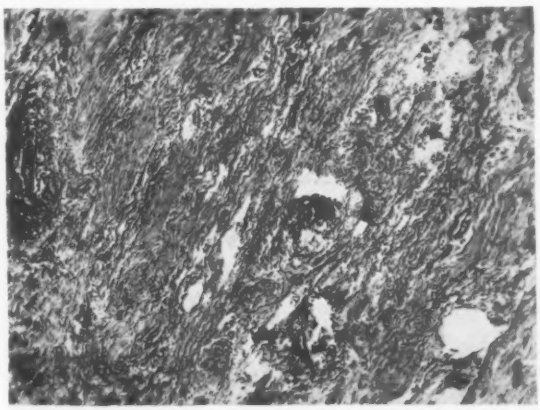

Fig. 6. Case 47. Photomicrograph of tissue from breast removed eleven months following treatment by roentgen radiation and interstitial radium. Much fibrosis is present. The debris is probably at the sites of former nests of cancer. Edema is seen, but no cancer cells can be found. Magnification 85×.

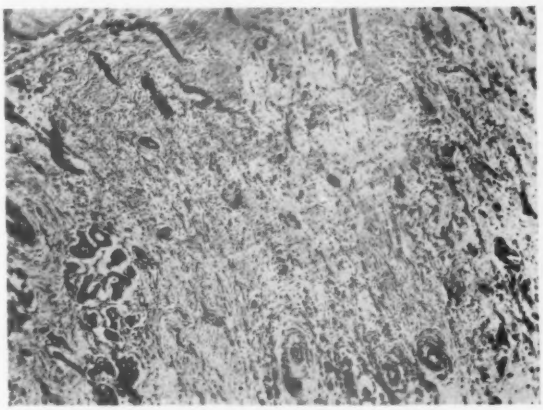

Fig. 7. Case 53. Photomicrograph of tissue from breast removed five months following treatment by roentgen rays and interstitial radium. Much fibrosis is present, but nests and strands of damaged cancer cells are also seen. Magnification 85×.

If the tumor has been excised just prior to insertion of the radium needles, it is frequently found that healing of the incision is delayed. The same is true of the incision through which a biopsy specimen has been removed.

If surgical procedures are carried out after interstitial radium treatment, it is most important that the skin edges be approximated without tension, since tissues which have been subjected to such intense irradiation heal more slowly and less securely than do normal tissues. In some of the patients who have had either a simple mastectomy or an attempted radical mastectomy, the healing has been very slow. If one case a simple mastectomy incision opened three weeks postoperatively. The wound later became necrotic and eventually caused the patient's death. Taylor[46] states that in his group of cases surgical wounds in the irradiated areas were very sluggish and seemed to be especially subject to infection.

ACCIDENTS

In placing the radium needles, puncture of the pleural cavity may easily occur. This is thought to have occurred in one of our cases. The needle was immediately withdrawn, and no harm resulted. Batchelor[3] states that one of his patients developed a

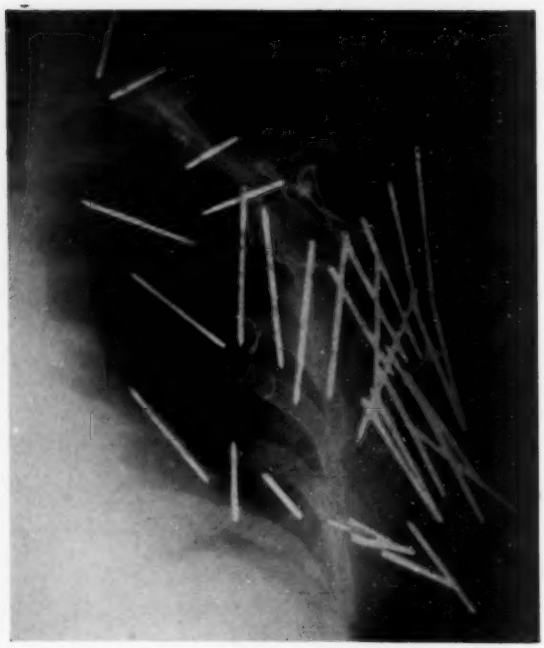

Fig. 8. Roentgenogram showing distribution of radium needles, except that the intercostal spaces have not been implanted. Needles extending toward the epigastrium have been omitted.

hemothorax which he thought resulted from puncture of the pleura by a needle inserted in the supraclavicular area. Introduction of needles in the supraclavicular region is always preceded by separation of the tissues beneath the skin punctures with a blunt hemostat for a distance of 15 mm. Injury to the blood vessels of the neck has not been observed. The axillary implantation is the most difficult, but if the arm is held in abduction and the needles are inserted without force and parallel to the blood vessels, no injury will result. Axillary injuries have not been recognized in the group of cases reported here.

At first, waxed silk was used to thread the radium needles. During removal of the needles from one of the first patients treated, this thread broke, necessitating an incision for recovery of a needle, with a considerable amount of resultant trauma. Other authors have reported similar experiences. In all of the patients treated more recently at this hospital, flexible monel metal wire has been used for threading the needles and has been found very satisfactory.

COMPLICATIONS

In the patient whose breast had to be incised for recovery of a radium needle, carcinoma appeared in several of the scars resulting from introduction of the needles. In one of Taylor's patients, implants of cancerous nodules occurred in relation to the skin punctures. Romanis[39] mentions a similar occurrence, and Keynes noted the development of an implant in an incision through which a specimen had been removed for biopsy.

DEVIATIONS FROM KEYNES' TECHNIQUE

Deviations from the technique originally outlined by Keynes have been:

1. The intercostal needles have been omitted in many cases unless the cancer was located in the inner hemisphere or in the nipple zone (Fig. 8).

2. Roentgen irradiation was given before radium implantation in 24 cases. This does not take into account 5 patients who had received a large amount of roentgen radiation over a long period of time before coming to this hospital. These 5 patients were all advanced cases, and only one of them is now alive. The roentgen treatments were all given in 1936 and 1937. The majority of these 24 cases belong in Group III. The local disease has been controlled in 18 and has remained uncontrolled in 6 of these patients.

3. In 22 cases, the tumor has been excised at the time of radium implantation. It is interesting to note that in none of these has there been a local recurrence. However, in many, too little time has elapsed since treatment to justify definite conclusions.

4. In 7 cases, a simple mastectomy has been performed and the axilla implanted. These were all cases with extensive involvement, and in none has a local recurrence developed. In one case the axillary disease has been controlled, while in 5 it has not been controlled. The seventh is a recent case and the only one of this group now living.

5. All of the platinum needles used have a wall thickness of 0.8 mm., whereas the needles used by Keynes have a wall thickness of 0.5 mm.

In 20 cases, biopsy specimens were taken, but the primary tumor was not removed. Nearly all of these were patients with advanced disease. The local disease was controlled in 13 cases, and 5 of the patients are now living. Of the total of 68 patients treated, 37 are now living. Every patient reported as being alive has been examined within the last four months, with the exception of one who lives in a distant city and was last heard from in May, 1938, at which time metastasis to the pelvis was suspected.

In every case a piece of tissue has been excised for microscopic examination and, with one exception, these specimens have permitted positive diagnosis. In any condition which requires as extensive treatment as does cancer of the breast it is imperative that there be no doubt about the diagnosis. Those with wide experience in this field may be mistaken about the diagnosis of a breast tumor. One of the cases in the series reported by Taylor[46] was considered to have cancer, but biopsy proved the tumor to be a gumma. In nearly all our cases, the biopsy specimen has been removed with the high frequency knife. This is considered to be safer than excision with the scalpel. The Hoffman punch has been tried a number of times, but satisfactory specimens have rarely been obtained by its use. When the tumor is accessible, it seems better to remove it entirely and then implant the surrounding tissue, rather than cut into the tumor for a specimen. When this is done, the electrosurgical knife is always used.

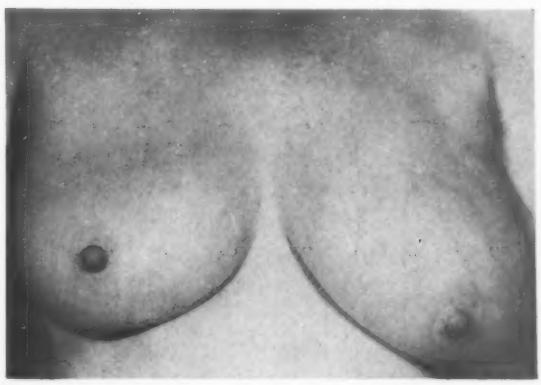

Fig. 9. Case 2. Tumor in upper inner quadrant, left breast. The tumor was excised and radium needles implanted.

Fig. 10. Case 2. Appearance of breast six years after radium irradiation.

CONTRAINDICATIONS

Patients with large, bulky breasts are unsuitable for interstitial radium treatment, since adequate irradiation cannot be secured. It is inadvisable to use this method if such distant metastases are present that the patient may not be expected to live long. The recumbency in bed during the period of irradiation is inadvisable for some elderly, feeble patients.

DISCUSSION

Recently a number of suggestions have been made to improve the method of placing the needles and retaining them in position. Grove and Holman[12] have devised a technique using long radon needles. A needle frame for holding trocars containing the radium has been devised by Brooke.[5] By this method, parallelism of the needles is assured. Hutchison[15] has altered the distribution of the needles and used some gold seeds. He now claims to obtain "radiologically complete" implantation. Other groups of patients have been treated by the simultaneous use of roentgen rays and in-

tersititial radium (Martin,[27] Melchart and Schloss[32]).

Regarding the amount of homogeneity obtained by this method, much difference of opinion exists. All are agreed that homogeneous irradiation is to be sought but is difficult to attain. Pack[33] believes that more diffuse irradiation is produced by long gold radon tubes than by gold seeds such as those used by Souttar.[45] Hutchison[15] admits that it is impossible to attain homogeneous irradiation, but he believes that there may be considerable latitude between the minimum and maximum amounts of radiation which may reach any given area. According to him, it is possible to obtain a minimum intensity throughout the entire area which will be lethal for tumor cells with a maximum intensity which will be insufficient to destroy the normal structures.

By the use of whole organ sections, Wainwright[47] has shown that the lump in the breast is only one focus of disease, and several other nodules may be present in the breast and even in the underlying muscle. For this reason, he believes that interstitial radium will fail in the treatment of cancer of the breast. Keynes has endeavored to overcome this by irradiating the entire breast and the lymphatic drainage areas. The criticism of Handley[13] is of the same order. He visualizes a carcinoma of the breast as consisting of a lump in the breast and perhaps another in the axilla, but more important, he believes, is a "widespread reticulum of microscopically permeated lymphatics, the limits of which cannot be determined by any clinical methods of observation."

Is there any evidence that the implantation of radium for cancer of the breast produces metastasis? Regaud[38] formerly advocated radium puncture, but in 1929 he expressed himself as being opposed to it on the ground that trauma to an organ containing cancer in which the cancer ordinarily metastasizes so easily is a dangerous procedure. Webster[49] observed several patients in whom rapid local and generalized metastases followed the insertion of interstitial radium. Pierquin and Richard,[36] as well as Simpson and Breed,[42] are of the opinion that metastasis is produced by implantation. It appears to Quick[37] that the trauma incident to the introduction and removal of radium needles is not included in the generally accepted idea of trauma in a tumor-bearing area. Lee, Pack,[25] and their associates have had wide experience with interstitial irradiation but have not observed dissemination of cancer as a result of this treatment. They believe that preliminary external irradiation lessens this possibility. Rubens-Duval[40] thinks that implantation is no more dangerous in this respect than surgical intervention. In the series of cases observed at this hospital, nothing has been seen to indicate that metastases have been produced by interstitial radium treatment.

CONCLUSIONS

The advantages of interstitial radium irradiation in treatment of cancer of the breast are:

1. The breast or a portion of it may be saved, and the patient spared a mutilating operation.
2. The risk of the procedure is probably less than that of radical breast amputation.
3. By this method it is possible to destroy the disease in some inoperable cases.
4. The method may be used for treatment of patients who refuse to consent to a radical operation.
5. If necessary, the treatment may be repeated.

The disadvantages of the method are:

1. It is not possible to implant large, bulky breasts and axillae in such a manner that anything approaching homogeneous irradiation will be secured.
2. It is necessary for patients receiving interstitial irradiation to remain in the hospital longer, and the period of convalescence is longer than in cases having had radical mastectomy.
3. Irradiation fibrosis frequently causes marked limitation of arm movement.

4. Closer follow-up is necessary for the detection of recurrences, and the interpretation of results is more difficult than in cases where the treatment has been by surgery.

5. The retraction of the breast and the telangiectases may be more disfiguring than an operative scar.

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ADENOCARCINOMA, SCIRRHOUS TYPE, OF THE LEFT MALE BREAST, WITH GENERALIZED BONE METASTASIS

CASE REPORT

By S. K. LIVINGSTON, M.D., F.A.C.S.
HINES, ILLINOIS

GESCHICKTER and Copeland,[1] in a series of 100 cases of adenocarcinoma of the breast, reported only one case in a male with generalized bone metastasis.

Because of the relative rareness of generalized bone metastasis in adenocarcinoma of the male breast, the following case is reported.

CASE REPORT

S.C.B. (Reg. No. 76010), a white male, aged forty-five, occupation dairyman, was admitted as a terminal case on March 6, 1937.

Chief Complaint. Pain in hips and chest, and weakness.

Family History. Negative.

Past History. Influenza in 1918.

Present Illness. Onset August, 1936, when pain and weakness developed in both legs.

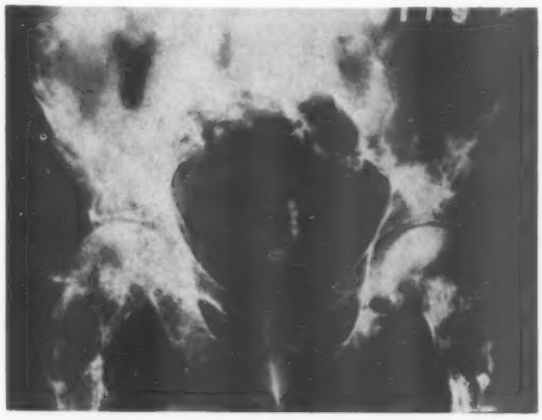

FIG. 1. Roentgenogram of pelvis showing extensive destruction suggestive of metastasis.

Routine examination on January 8, 1937, revealed a small tumor in the left breast. A biopsy report showed scirrhous adenocarcinoma, and a radical mastectomy was done in January, 1937. Roentgen examination on January 22, 1937, revealed no evidence of bone metastasis. On examination March 6, 1937, the patient was cachectic and emaciated. There was a healed postoperative scar from a radical left mastectomy. No lymphadenopathy or abdominal masses were palpable. There was slight atrophy of the musculature of the left thigh.

FIG. 2. Roentgenogram of humerus showing extensive destruction suggestive of metastasis.

Roentgen Findings. Roentgenographic examination shows extensive destruction of the pelvis, lumbar spine, ribs, humerus, as well as a portion of the scapula and clavicle. The appearance is that of extensive metastasis.

Histological Report, Slide No. 14093. A neoplasm composed of small masses of epithelial cells supported by fibrous connective tissue

[1] Geschickter, C. F., and Copeland, M. M. Bone Tumors. American Journal of Cancer, New York.

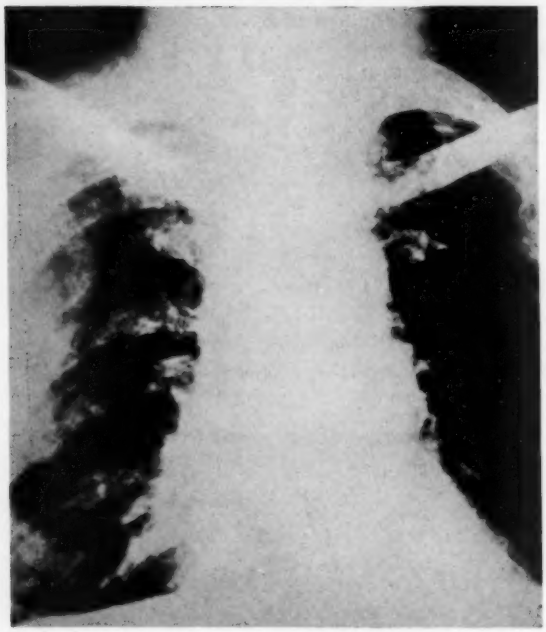

Fig. 3. Roentgenogram of ribs showing extensive destruction suggestive of metastasis.

stroma. In one area there appears a tendency toward the formation of acini; however, in the greater portion of the section there is a predominance of fibrous stroma producing a pseudo-alveolar structure enclosing small groups of cells. Mitotic figures are noted.

Diagnosis. Adenocarcinoma, scirrhous type, eft breast, with extensive metastasis to bone.

Treatment. Symptomatic. The patient was a terminal case during the entire period of hospitalization and died on June 17, 1937. Post-mortem examination confirmed the above find-

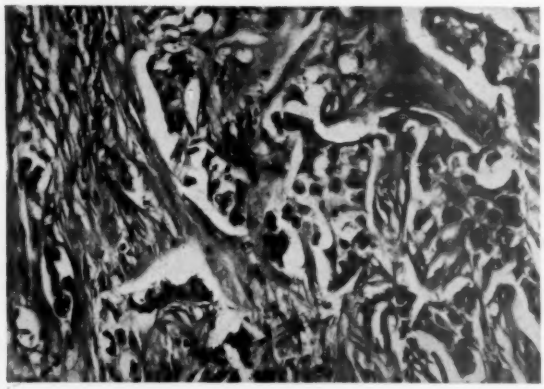

Fig. 4. Photomicrograph of specimen from left breast showing a neoplasm composed of small masses of epithelial cells supported by fibrous connective tissue stroma with some tendency toward the formation of acini. There is a predominance of fibrous stroma producing a pseudo-alveolar structure enclosing small groups of cells. Mitotic figures are noted.

ings and added metastasis to spleen, liver, pleura.

Conclusion. A case of extensive bone metastasis from adenocarcinoma, scirrhous type, in a male breast, is reported.

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THE ROENTGEN TREATMENT OF LOBAR PNEUMONIA

By EMMETT B. SETTLE, M.D.
ROCK PORT, MISSOURI

ROENTGEN therapy has a definite place in the modern armamentarium for the treatment of lobar pneumonia. The purpose of this paper is to add our experience in treating a well controlled group of cases by that method to the few scattered reports appearing in recent literature.

The favorable influence of roentgen rays in the treatment of unresolved pneumonia was first described by Musser and Edsall as early as 1905. Since that time, occasional reports have appeared describing varying degrees of success in the roentgen treatment of unresolved and acute pneumonias. However, the heterogeneity of pneumonias treated, the absence of any well controlled series of cases, and the wide variations in the technical factors employed, made any critical evaluation of the results extremely difficult.

Fried, in 1937, produced hepatizing pneumonia in guinea pigs, pathologically resembling lobar pneumonia, which he successfully treated by roentgen therapy. He concluded this treatment was beneficial because grossly the lungs of the control animals showed complete hepatization, while the lungs of the irradiated animals appeared almost normal with only scattered areas of pneumonia; likewise, microscopically, the irradiated lungs showed less congestion, edema, infiltration and abscess formation than the non-treated.

Powell has treated lobar pneumonia by roentgen therapy with very satisfactory results, so much so that he believes this type of treatment should be the method of choice in this disease. The cases in his well controlled series, reported from time to time over the past three years, numbered 231 in February, 1939, with the low mortality rate of 7 per cent.

Solis-Cohen and Levine, following Powell's technique, produced comparable results in their series of 42 cases of lobar pneumonia.

The material presented in this paper was taken from a group of 34 proved cases of lobar pneumonia, unselected and consecutively admitted to the Medical Service of the St. Francis Hospital during the 1938–1939 season, each case presenting roentgenographic and bacteriological proof of the diagnosis. Roentgen treatment was employed in every case, none of the so-called specific drugs or sera being used, and the only therapeutic agents other than roentgen radiation consisting of opiates, various barbituric sedatives, oxygen and parenteral fluids as indicated.

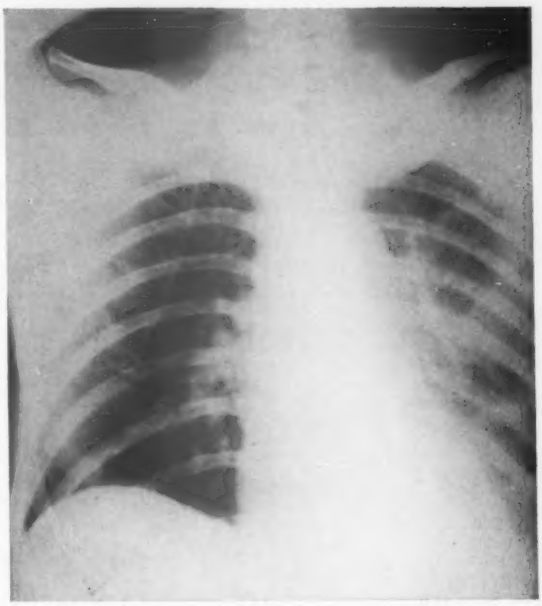

FIG. 1. Case 1. Pneumonic consolidation of left lower lobe. Fourth day.

The clinical response to this therapeutic regimen is well illustrated by the following cases, which were chosen from the group as typical of the various reactions observed.

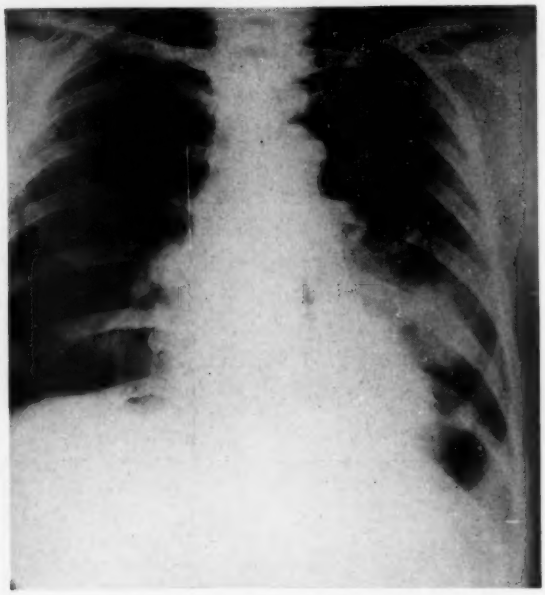

Fig. 2. Case 1. Roentgenogram on ninth day shows partial resolution of pneumonic process.

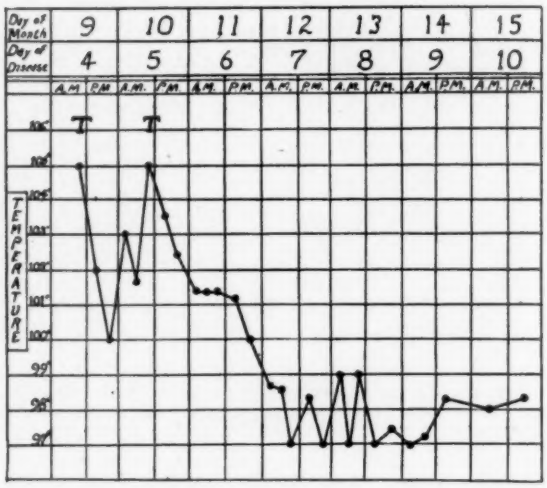

Fig. 3. Case 1. Reproduction of fever chart with roentgen therapy indicated at "T."

REPORT OF CASES

Case 1. G. A. G., male, aged sixty, realtor, was admitted on January 9, 1939, complaining of chills, fever, cough, and pain in left chest. Symptoms progressively worse over past three days. Examination: Temperature 105° F. Marked cyanosis and dehydration. Signs of consolidation over entire left chest. Chest roentgenogram revealed diffuse consolidation of left lower lobe. White blood cells 19,000. Sputum examination yielded staphylococci predominantly. Course: Roentgen treatment on admission and repeated on second day. Oxygen by nasal catheter for first forty-eight hours and parenteral fluids as indicated. Marked clinical improvement by third day with temperature dropping to normal. Rusty sputum continued to show predominantly staphylococci and yielded *Staphylococcus aureus* on culture. Discharged on January 22, 1939 (Figs. 1, 2 and 3).

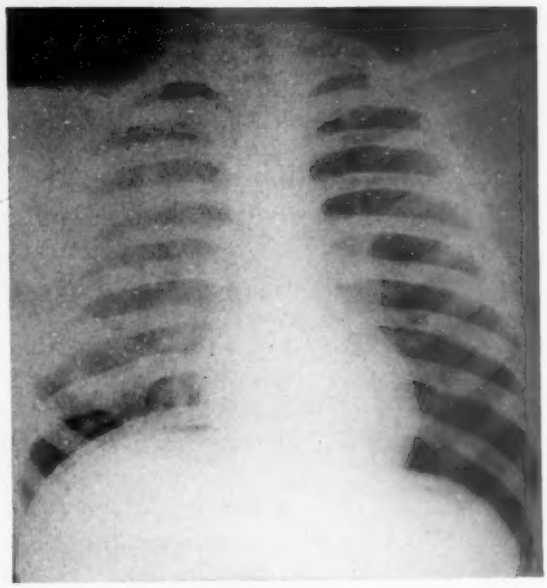

Fig. 4. Case 11. Pneumonic consolidation of right lower and middle lobes. Sixth day.

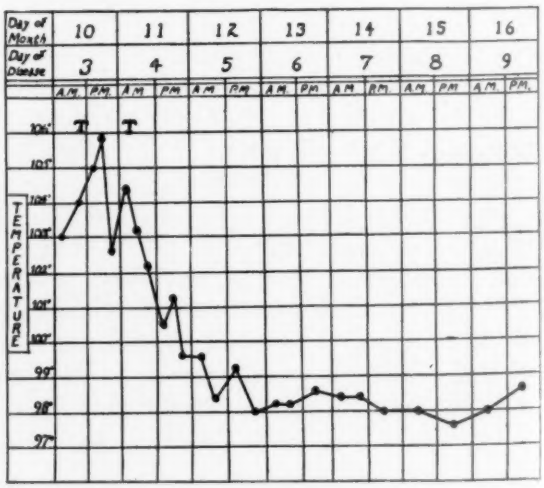

Fig. 5. Case 11. Reproduction of fever chart with roentgen therapy indicated at "T."

Case II. G. P., male, aged twenty, college student, was admitted on January 10, 1939, complaining of chills, fever and pain in right chest of two days' duration. He had had a cold for the previous week. Examination: Temperature 105° F. Physical signs indicative of consolidation of the right middle and lower lobes. White blood cells 29,000. Course: Roentgen treatment on admission and repeated on second day. Marked improvement by third day with temperature receding to normal. Rusty sputum on third day yielded pneumococcus Type II. Chest roentgenogram on fourth day revealed consolidation of right lower and middle lobes with beginning resolution. Uneventful convalescence. Discharged on January 20, 1939 (Figs. 4 and 5).

Case III. M. H., aged eleven, school girl, was admitted on April 18, 1939, complaining of fever, chills and pain in the chest for the past eighteen hours. She had had a cold for three days previously. Examination: Temperature 103.6° F. Physical signs of consolidation in right lung. Chest film revealed diffuse consolidation of right middle lobe. White blood cells 27,700. Sputum examination yielded pneumococcus Type I. Course: Roentgen treatment on admission. Marked improvement by second day. White blood cells 16,100 on second day. Temperature elevation to 101° F. the second day but normal thereafter. Uneventful convalescence. Discharged on April 24, 1939 (Figs. 6, 7 and 8).

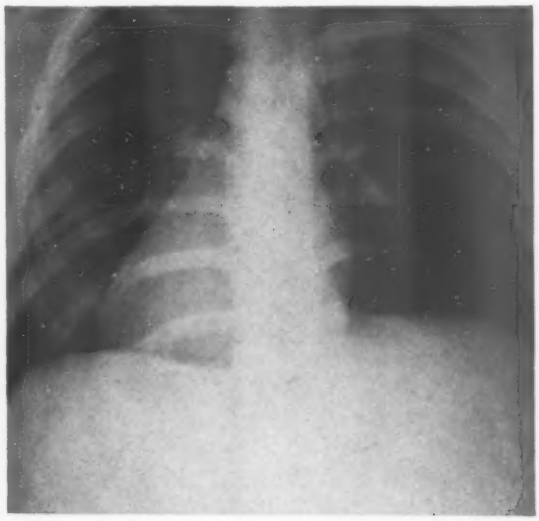

Fig. 6. Case III. Early pneumonic consolidation of right middle lobe.

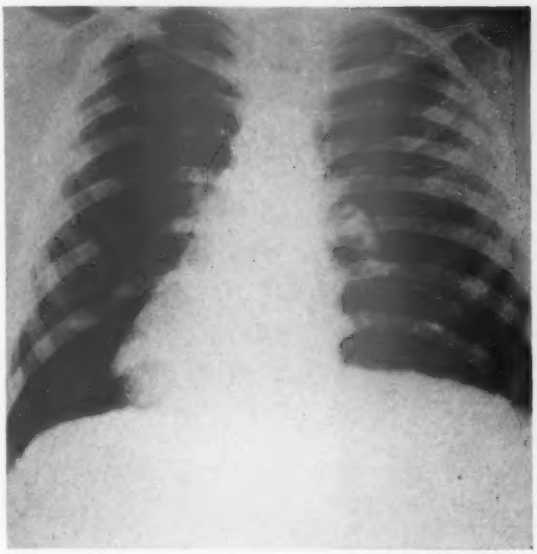

Fig. 7. Case III. Roentgenogram on eleventh day shows only partial resolution of the pneumonic process.

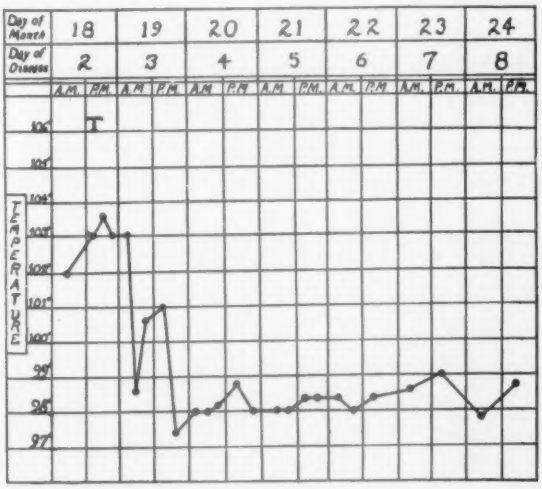

Fig. 8. Case III. Reproduction of fever chart with roentgen therapy indicated at "T."

Case IV. O. S., male, aged forty-four, farmer, was admitted on February 6, 1939, complaining of chest pain and fever for the past three days. Examination: Temperature 104° F. Physical signs indicated a complete consolidation of the left lung. Chest films revealed diffuse consolidation of the left lower lobe. White blood cells 11,000. Sputum examination yielded pneumococcus Group IV. Course: Roentgen treatment on admission and repeated on second day, followed by clinical improvement, fall in tempera-

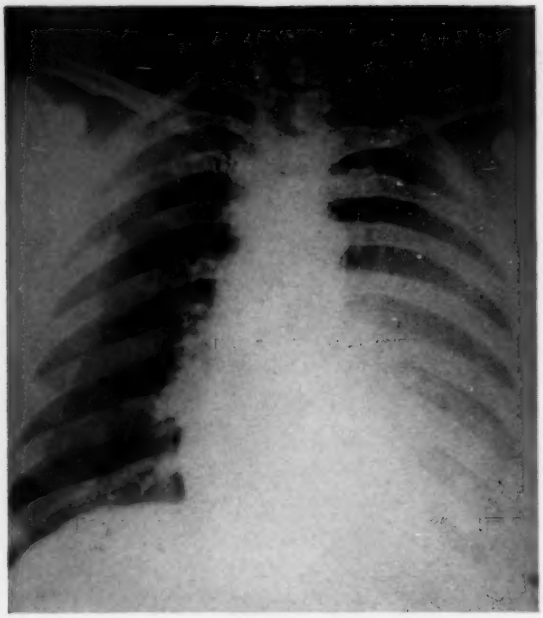

Fig. 9. Case iv. Pneumonic consolidation of left lower lobe. Fourth day.

ture to normal, and an uneventful convalescence. Discharged on February 15, 1939 (Figs. 9, 10 and 11).

Case v. B. M. H., male, aged twenty-one months, was admitted on January 20, 1939,

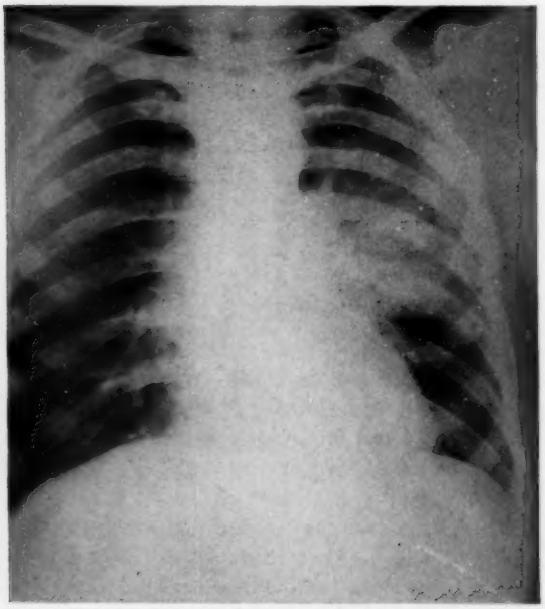

Fig. 10. Case iv. Roentgenogram on fourteenth day shows partial resolution of the pneumonic process.

with complaint of chills, fever and dyspnea for three days. Had had pertussis several weeks before which culminated in "bronchopneumonia" one month ago. However had been well for two weeks prior to present illness. Examination: Temperature 105° F. Marked toxicity and dehydration. Acute otitis media, right; congestive stage. Physical signs of consolidation over entire right lung. Meningismus Grade 3. Chest film revealed consolidation of right middle and upper lobes. Sputum examination yielded pneumococcus Type 1. White blood cells 30,700. Course: Roentgen treatment given on admis-

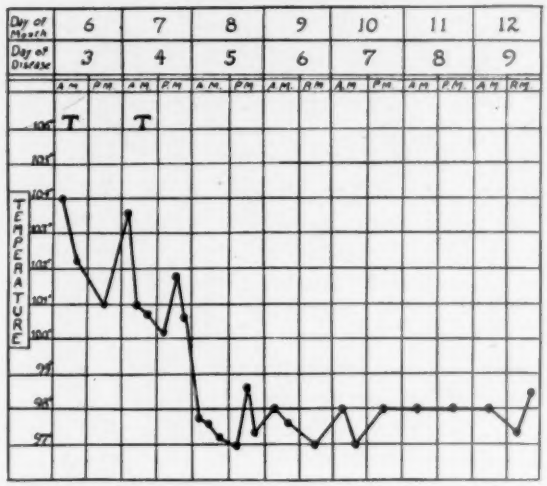

Fig. 11. Case iv. Reproduction of fever chart with roentgen therapy indicated at "T."

sion and repeated on second day. Oxygen and parenteral fluids as indicated. Followed by marked clinical improvement and temperature fall by lysis. Roentgen treatment repeated on fifth day because of persistent fever and leukocytosis to 22,800. Followed by fall in temperature to normal and rapid convalescence. Incidentally, acute otitis media was treated by roentgen therapy and promptly subsided. Discharged on February 2, 1939 (Figs. 12, 13 and 14).

Case vi. C. M., aged eight, school boy, was admitted on March 5, 1939, complaining of generalized abdominal pain for the past twenty-four hours. No nausea or vomiting. He had had a severe cold for several days prior to admission. Examination: Temperature 103° F. Physical signs gave evidence of consolidation in left lower lobe. Chest films revealed an area of increased density, presumably consolidation, in

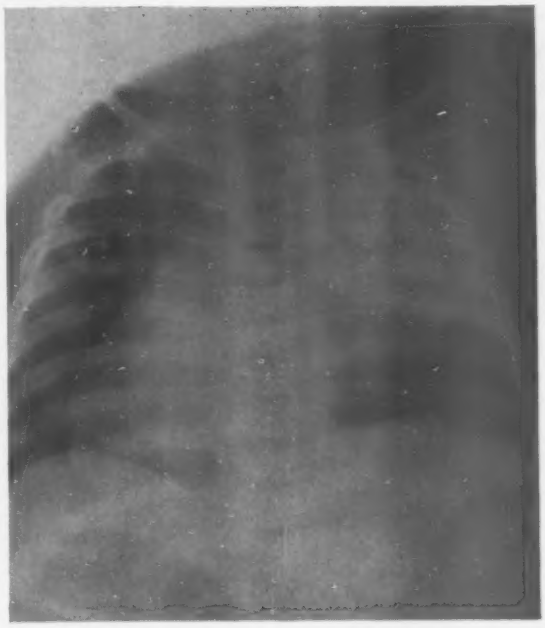

Fig. 12. Case v. Pneumonic consolidation of right middle and upper lobes. Fourth day.

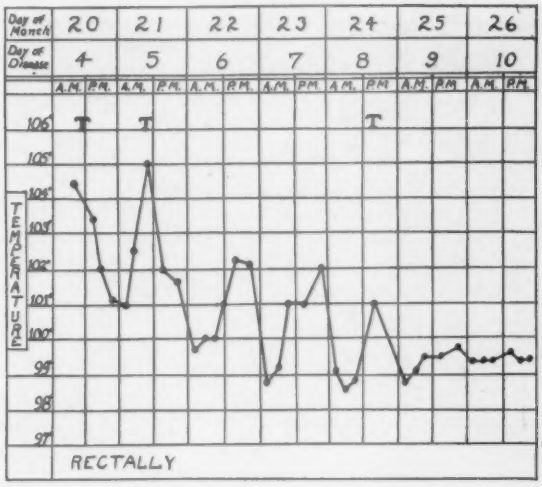

Fig. 14. Case v. Reproduction of fever chart with roentgen therapy indicated at "T."

the left lower lobe. White blood cells 30,400. Sputum examination yielded pneumococcus Type II. Course: Roentgen treatment on day of admission and repeated on second day, followed by marked clinical improvement and drop in temperature to normal. White blood cells 21,000 decreasing to 15,000 on second and third days respectively. Chest films on sixth day after first treatment indicated beginning resolution. Uneventful convalescence. Discharged on March 12, 1939 (Figs. 15, 16 and 17).

Case vii. F. B., male, aged forty-four, farmer, was admitted on February 17, 1939, complaining of fever, pain in right chest, cough and bloody sputum. He had had "pneumonia" for a week prior to admission but had had no treatment. During the war he had been gassed and suffered a lung injury. Had been in poor health the past few months. Examination: Temperature 105° F. A badly dehydrated, cyanotic, restless patient in extremis. Physical

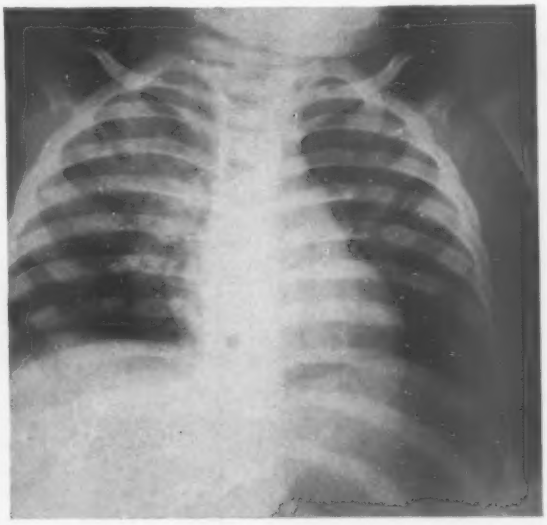

Fig. 13. Case v. Roentgenogram on fifteenth day shows partial resolution of the pneumonic process.

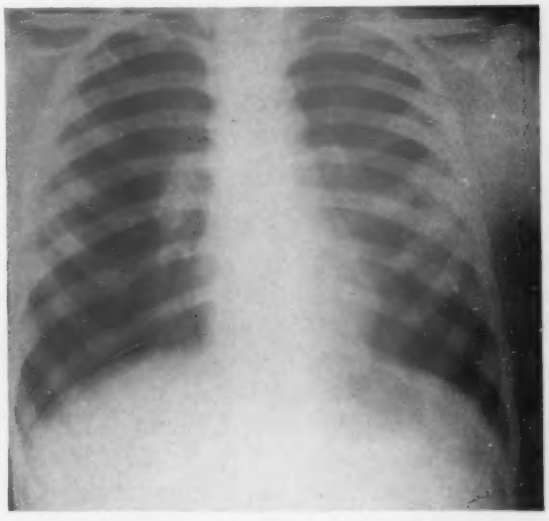

Fig. 15. Case vi. Pneumonic consolidation of left lower lobe. Third day.

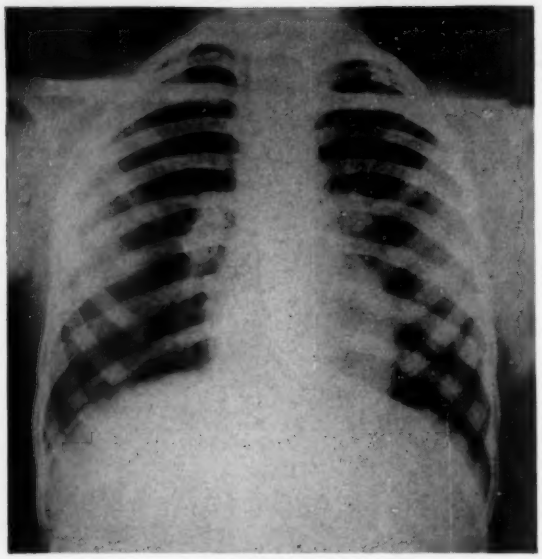

Fig. 16. Case vi. Roentgenogram on eighth day shows partial resolution of the pneumonic process.

signs of consolidation over right lower lobe. Chest film revealed dense consolidation of right lower and middle lobes. White blood cells 17,000. Sputum examination yielded pneumococcus Type iii. Course: Roentgen treatment on admission and repeated following day but followed by no improvement. Became increasingly restless, apprehensive, cyanotic and dyspneic. He developed terrific pulmonary edema following cardiac collapse and died on the fourth day in spite of transfusions, oxygen and supportive treatment. Autopsy: Typical lobar pneumonia of the entire right lung in the stage of gray hepatization (Figs. 18, 19 and 20).

CLINICAL RESPONSE

The favorable response to roentgen treatment, as illustrated by the above cases, was characterized by relief of pain, fall in temperature, dramatic improvement in the patients' general well-being, decrease in leukocytosis and resolution of consolidation.

The relief of pain, for which patients were extremely grateful, usually was noticed within six to twelve hours after the first treatment and was the first indication of improvement, usually occurring prior to the fall in temperature. As the pain was relieved, the dyspnea and anxiety lessened and patients were much more comfortable.

In Case i, the patient's severe pain was relieved within twelve hours after his first treatment, although the temperature did not break to normal until thirty-six hours later after a second treatment.

The fall in temperature occurred usually in twelve to thirty-six hours and was of the crisis type. This is graphically illustrated in Case ii where the temperature fell from 106° F. to normal on the fourth day of the illness, thirty-six hours after the first treatment; and in Case iii where the temperature fell from 103.6° F. to normal on the third day of the illness, twenty-four hours after treatment. Occasionally the temperature fall was of the lysis type. Case v presents this picture, having shown marked clinical improvement after the second treatment, but continuing a low grade pyrexia which persisted until a third treatment was administered on the fifth hospital day. In Case vii the roentgen treatment was followed by depression of the fever, but no improvement in the patient's condition which terminated fatally.

The recession in temperature was accompanied by a dramatic improvement in the patients' general well-being. An extremely ill and anxious patient before treatment was often comfortable and resting quietly within twelve to twenty-four hours thereafter.

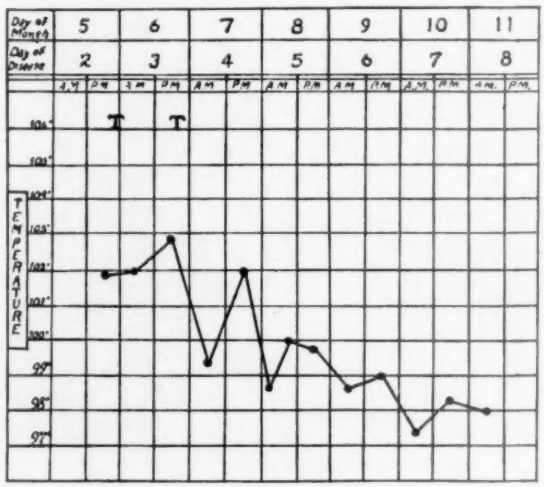

Fig. 17. Case vi. Reproduction of fever chart with roentgen therapy indicated at "T."

The initial decrease in leukocytosis occurred generally within a few hours after the fall in temperature, following which the leukocyte count gradually returned to normal.

Resolution of consolidated lung tissue, as studied roentgenographically, did not occur until several days after the clinical response. Frequently, in an afebrile patient several days' convalescent, a considerable area of consolidation was present as visualized by roentgenographic examination. Autopsy in Case VII revealed a diffuse consolidation of the entire right lung. The gross and microscopic picture was typical of lobar pneumonia in the stage of gray hepatization and no changes, gross or microscopic, attributable to roentgen therapy, were present (Fig. 20).

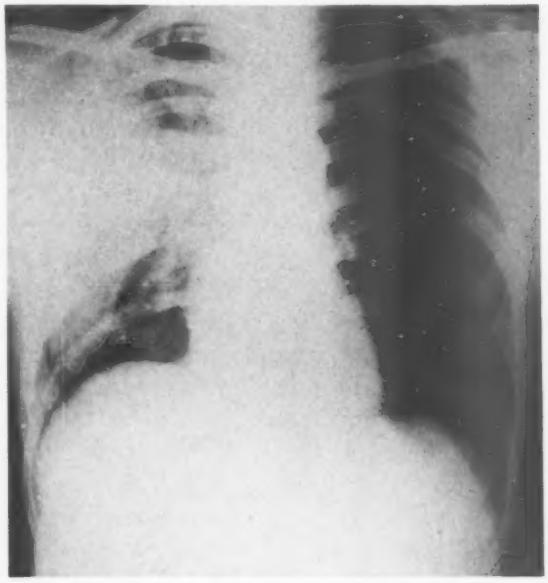

FIG. 18. Case VII. Pneumonic consolidation of right lower and middle lobes. Seventh day.

In the 2 fatal cases, as in Solis-Cohen and Levine's experience, no improvement was shown at any time during the treatment. A poor prognosis is indicated if the favorable response does not occur by the fourth day.

STATISTICAL RESULTS

The statistical results are shown in Table 1. A total number of 34 cases were treated

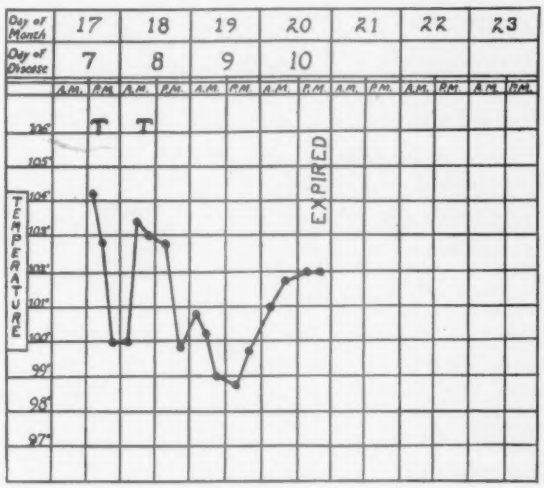

FIG. 19. Case VII. Reproduction of fever chart with roentgen therapy indicated at "T."

of which there were 32 recoveries and 2 deaths, or a mortality rate of 6.2 per cent. It is interesting to note that in one case pneumococci were not isolated from the sputum yet this patient presented typical clinical and roentgenographic evidence of lobar pneumonia and reacted favorably to treatment. Powell has previously pointed out the obvious value of roentgen therapy in those cases where type specific pneumococci are not isolated.

There were remarkably few complications. Positive blood cultures were obtained from each of the 2 fatal cases (one Type I, one Type III), both of which were in the

FIG. 20. Case VII. Microscopic sections of the consolidated lung show only the usual histopathology of lobar pneumonia in the stage of gray hepatization. Roentgen therapy did not alter the usual gross or histopathological findings.

seventh day of the disease and practically moribund on admission.

Five cases, or 15 per cent, required only one treatment, while 75 per cent received two treatments. Of the former group, all were admitted on or before the second day of the illness, while in the latter only 3 were admitted at this early stage. Early treatment is extremely important since, in general, it not only necessitates fewer treatments, but also results in an earlier occurrence of the favorable response.

TABLE I

STATISTICAL RESULTS

Total number of cases treated	34
Recovered	32
Died	2
Mortality rate	6.2%
Complications	4
Otitis media (recovered)	1
Unresolved pneumonia (recovered)	1
Bacteriemia (died)	2
Bacteriological classification	
Pneumococcus Type I	7
Pneumococcus Type II	14
Pneumococcus Type III	4
Pneumococcus Group IV	8
Staphylococcus aureus	1
Number of roentgen treatments	
One treatment	5
Two treatments	25
Three treatments	4
Average hospital days per case	11.2

METHOD OF TREATMENT

The therapeutic regimen uniformly adopted consisted of early and adequate roentgen therapy plus the general supportive measures and nursing care usually utilized in the treatment of pneumonia.

The first roentgen treatment was given as soon as possible after the diagnosis was made. Suspected cases were admitted directly to the roentgen department where chest roentgenograms were made and material for laboratory studies obtained. Roentgen treatment was then given before moving the patient to the ward and was repeated in twenty-four hours if the favorable response had not occurred within that time. A third treatment was given on the third or fourth day where indicated.

All treatments were given in the roentgen department, and in our experience a very sick patient was unharmed and but little inconvenienced by being warmly wrapped, carefully transferred to a wheel litter and conveyed from one floor to another.

The usual supportive and nursing measures employed in the treatment of pneumonia were emphasized and carefully followed.

TECHNIQUE

The following technical factors were uniformly employed and are similar to those recommended by Powell: Potential 130 kv. (peak); current, 5 ma.; anode-skin distance, 30 cm.; filter, 3 mm. Al; average dosage, 200 r at each treatment through one field for adults, 150 r for children. That portal was used which offered the best approach to the involved area. In general, the anterior field was irradiated at the first sitting, the posterior and lateral portals at the second and third sittings. However, we have more recently employed the axillary or lateral portal at the first sitting with somewhat better results. Anatomically, this route offers the most direct approach to the greatest area of lung tissue.

SUMMARY

Roentgen therapy was employed to the exclusion of specific drugs or sera in the treatment of 34 cases of lobar pneumonia with a resulting mortality rate of 6.2 per cent. Roentgen treatment produces a typical clinical reaction in lobar pneumonia, characterized by relief of pain, fall in temperature, dramatic improvement in the patient's general well being, decrease in leukocytosis and finally resolution of consolidation.

An explanation as to why this response occurs or as to the physiologic effect of roentgen therapy in acute infections is beyond this paper's scope or my knowledge. The several theories thus far presented lack adequate scientific proof. Many of the

depths of the question of immunity itself remain unplumbed. However, many drugs whose complete physiologic action is vaguely if at all understood are standard stock in every dispensary. So the place of roentgen therapy in the treatment of lobar pneumonia or of acute infections in general should not be minimized or discarded simply because the complex questions of physiology and immunity involved are poorly understood.

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EXPERIMENTAL STUDIES WITH "CONTACT" ROENTGEN RAYS

THE TIME-INTENSITY FACTOR OF THE "TUMOR DOSE" FOR RAT SARCOMA 39 IN SITU

By J. GERSHON-COHEN, M. D., HARRY SHAY, M. D., *and* S. S. FELS, LL. D.
PHILADELPHIA, PENNSYLVANIA

MUCH has been written about the time-intensity factor in radiotherapy, but the fact that clinical radiologists swing from one system of time-intensity regime to another so readily is in itself an indication of the lack of conviction concerning the superior efficiency of any one method. Whether to use high intensity-short time, low intensity-long time, or to grade these factors up or down; whether to use the Pfahler saturation technique or the Coutard method, continues to puzzle the active radiologist.

Does contact roentgen therapy differ from other forms of roentgen therapy for superficial cancer in its biologic action, and does a change in the timing of fractional doses have a bearing? To answer these questions, the experiments here reported were arranged to follow as closely as possible the techniques of the clinical radiologist, and at the same time, to get away from the methods of previous experimental approach in the study of the time-intensity factor. A review of the literature and comparison of results are postponed until the completion of several additional experiments now in progress.

In this series of investigations, the Crocker rat sarcoma 39 was selected as the malignant tumor because the degree of its radiosensitivity may be graded as average among transplantable tumors. The animals usually succumb during the seventh week after transplant (see Fig. 5). Because the radiologist seldom gets the opportunity to treat malignant tumors in their earliest stages, we selected that stage of tumor growth which corresponds to about the end of the first third of tumor life. All roentgen treatments were started from the fifteenth to the nineteenth day after transplant when the tumors averaged 10 to 15 mm. in diameter. Sarcoma 39 sometimes regresses spontaneously after short varying periods of growth, but in our stock of animals maintained in optimum health with standard well-regulated diets and in rooms with controlled regulation of temperature and humidity, spontaneous regression never exceeded 5 per cent.

The Chaoul contact apparatus was used in these experiments with the following factors: 60 kv., 0.12 mm. Cu filter, 3 cm. distance from the target to the center of the tumor obtained by slight pressure of the applicator against the skin around the tumor, the field 2 cm. in diameter, and 450 r/min. measured with a Siemens dosimeter checked by the National Bureau of Standards.

In one group of experiments, single doses of varying intensities ranging from 3,000 r to 15,000 r were given to the tumor. We found that as much as 15,000 r could be given before the animal succumbed from the effect of the treatment alone, and that 10,000 r caused almost uniform destruction of the tumor with survival of the animal. No recurrence appeared in any of these animals and the observations so far have extended nine months beyond treatment (Figs. 1, 2 and 3).

Doses of 6,000 r resulted in destruction of some tumors, roughly 60 per cent, and smaller doses of 3,300 r rarely destroyed the tumors, but often effected delay in the growth of many of them (Fig. 4). Because a dose of 10,000 r could be depended on to cause destruction of the tumor when given under the conditions noted above, this dose was selected to study the effect of the time factor.* Accordingly, groups of tumors were

* Dr. F. C. Wood, in a personal communication, informs us that 3,500 r applied to tissue cultures of rapidly growing animal tumors is lethal in a single dose. This is not to be confused with the effect of roentgen rays on these tumors in situ when conditions

given totals of 10,000 r divided into *daily* treatments and spread out equally so as to be given within seven, fourteen, twenty-eight, forty-two or seventy days. It might be noted here that our time factors are not limited to that involved with each single divided dose, but related to the much longer intervals *between* these divided doses. For this reason, our results are not to be confused with the mass of experimental work dealing with time-intensity factors which are limited to a single application of roentgen radiation.[3] Nor should

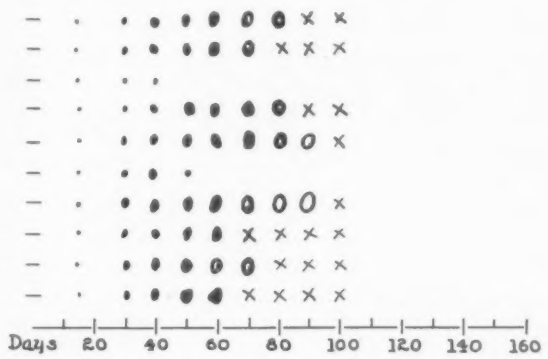

Fig. 1. The effect of 3,300 r in single dose of "contact" roentgen rays on sarcoma 39 given seventeen days after tumor transplant.

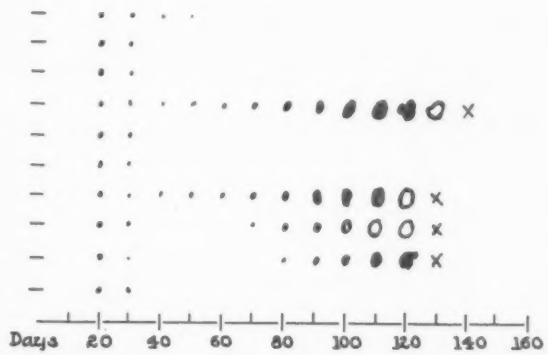

Fig. 2. The effect of 6,600 r in single dose of "contact" roentgen rays on sarcoma 39 eighteen days after tumor transplant.

the fact that these tumors were all transplanted subcutaneously be forgotten since our results might have been different with more deeply placed tumors. Russ and Scott[4] pointed out some time ago the relation between the roentgen-ray wave length and the depth of the tumor, but their observations do not bear on our work which is

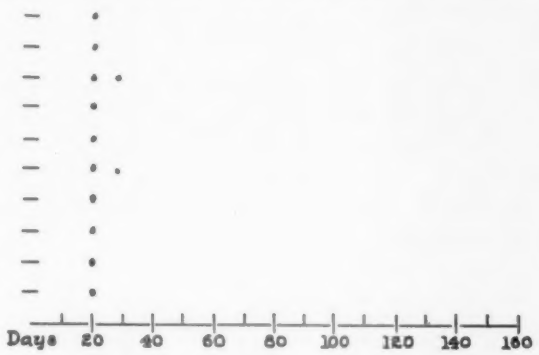

Fig. 3. The effect of 10,000 r in single dose of "contact" roentgen rays on sarcoma 39 given eighteen days after tumor transplant. All tumors disappeared within ten to twenty days.

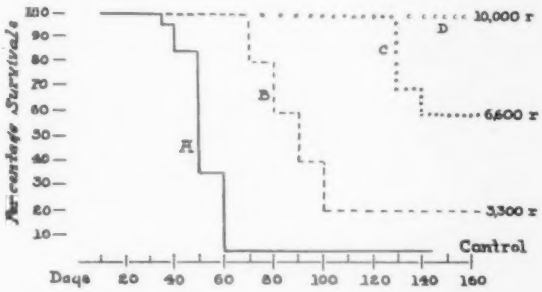

Fig. 4. *A*, survival periods curve of rats bearings arcoma 39 without treatment; *B*, of rats bearing sarcoma 39, treated eighteen days after transplant with a single dose of 3,300 r "contact" roentgen rays; *C*, of group treated as in *B* with single dose of 6,600 r; *D*, no deaths with uniform destruction of tumor in another group of rats treated with single dose of 10,000 r.

limited to relatively superficial tumors, the theoretical province for "contact" roentgen irradiation.

In general, our experiments revealed that as the doses decreased in size, and as time necessary to reach the final total dose increased, the destructive effect on the tumor decreased. Thus, when a dose of 10,000 r was given by one application, the tumor was completely destroyed. But when this dose was divided into six parts, and given on six successive days, only about 60 per cent of the animals survived with disappearance of the tumors (Fig. 9*B*). When

approach more nearly human tumors insofar as Dr. Wood believes that 10,000 to 12,000 r to the skin or 6,000 to 8,000 r in the tumor is necessary to get a lethal effect. This closely resembles our findings with sarcoma 39 in situ.

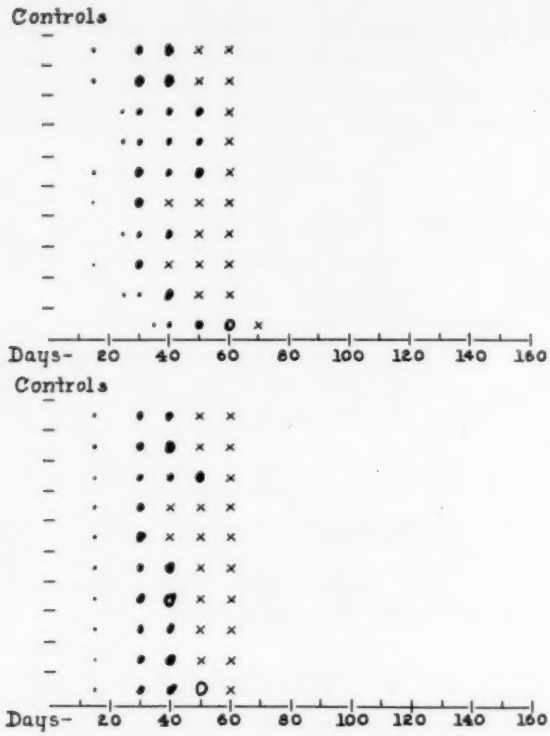

FIG. 5. Survival periods of Wistar rats after sarcoma 39 transplants (control series).

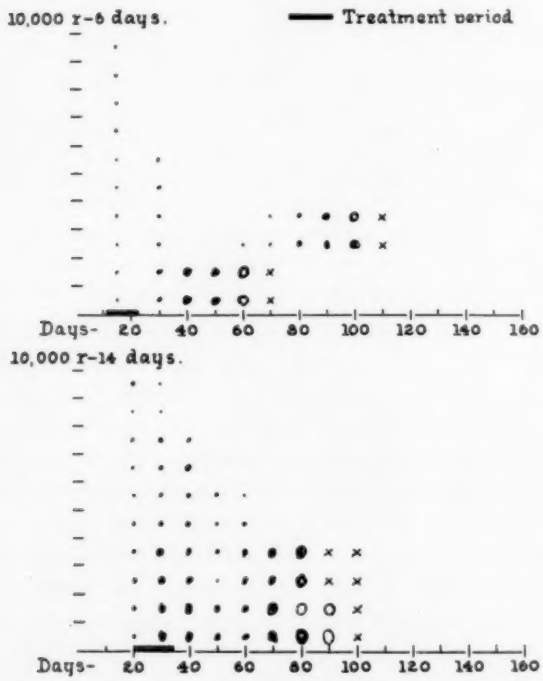

FIG. 6. Survival periods following administration of 10,000 r "contact" roentgen rays to sarcoma 39.

10,000 r was divided into twelve successive daily doses, while only 60 per cent of the tumors were destroyed, the longevity curve was slightly changed (Fig. 10*A*). When the divided doses finally were made so small

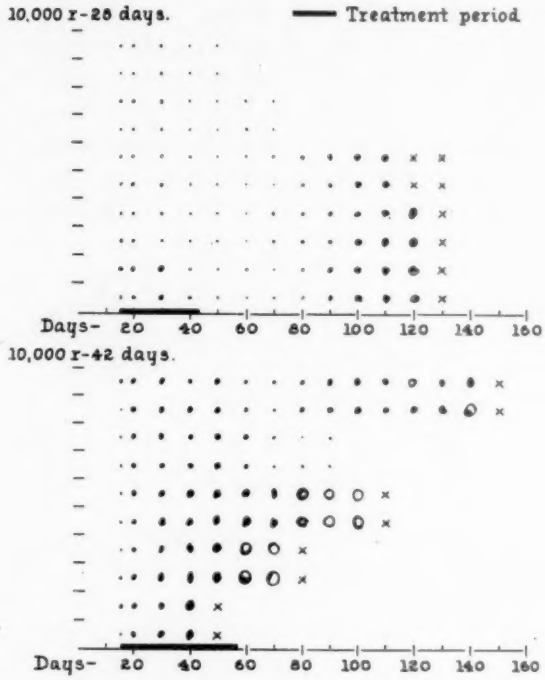

FIG. 7. Survival periods following 10,000 r "contact" roentgen rays given in equal parts within twenty-eight days and within forty-two days; treatment started nineteen days after tumor transplant.

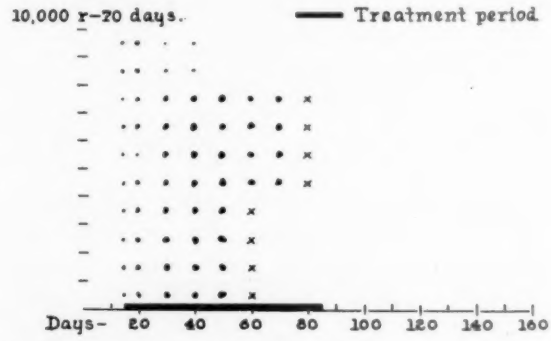

FIG. 8. Survival periods following 10,000 r, given 140 r daily extending to seventieth day when possible; treatment started fifteen days after tumor transplant.

The dose was divided into equal parts given within six days and within fourteen days; treatment started eighteen days after tumor transplant.

that it required seventy days to reach a total of 10,000 r, then *no* destructive effect on the tumor was evident (Fig. 11*B*). These findings are at variance with the impression of Morison and Adams[2] who believed that in humans "the divided dose, say 6,000 r in ten days produced a much greater effect than 6,000 r delivered in a single dose."

In the group in which a total of 10,000 r was given within twenty-eight days, approximately 350 r daily, the number of cured survivals was less than in those groups given the same total in shorter periods; but one notes from the longevity curves a more prolonged temporary good effect (Fig. 10*B*). Clinical radiologists are generally conscious of better results where the course of therapy is completed within four to six weeks, and it may be more than coincidence that our experimental findings seem to bear out these clinical impressions. Yet the final effect on the tumor indicates a progressive diminution as the time to reach the total dose increases. These experimental observations in vivo substantiate Canti's[1] work with tissue cultures in vitro. If the survival curves in Figure 7 can be explained by the law of Bergonié and Tribondeau that the cell is more radiosensitive during mitotic activity—the final results show that the clinical radiologist has a more advanced point of view when he feels that his only hope for eradicating a tumor by irradiation is to administer as many roentgens as necessary to destroy all the cells of a tumor, regardless of their phase of mitosis. If, however, it is necessary to increase the time factor, increases in the total dosages are necessary to get the optimum effect. This total dosage is not

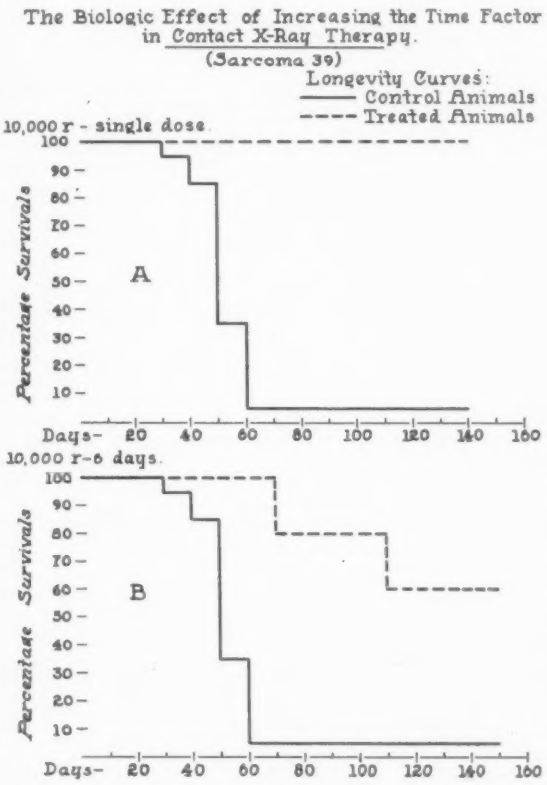

Fig. 9. *A*, comparative longevity curves of control rats and rats treated with sarcoma 39. The treated tumors were given 10,000 r "contact" roentgen rays in single dose. *B*, the longevity curves in which the treatment was given daily, six doses totaling 10,000 r.

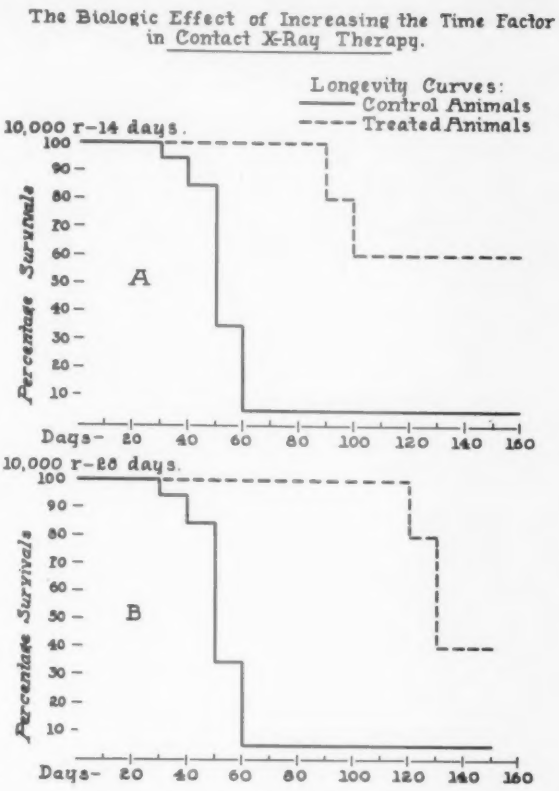

Fig. 10, *A*, a total of 10,000 r given in fourteen days; *B*, 10,000 r given in twenty-eight days.

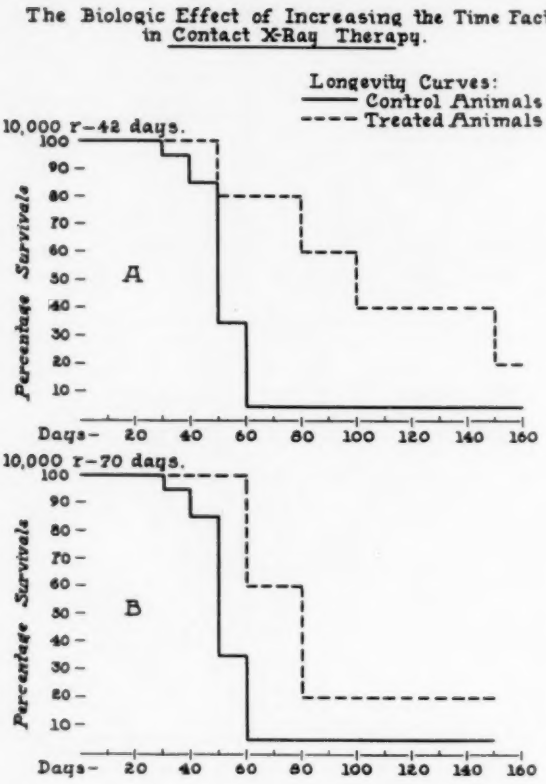

FIG. 11. *A*, 10,000 r given in forty-two days; *B*, 10,000 r given in seventy days. There is very little difference in the survival curves between the control and treated animals when too long a period of time is taken to deliver that dose which is lethal in a single application.

seriously affected by the wave lengths of the roentgen rays used, and our findings would substantiate Wood's[5] convictions when he said there is little difference between the biologic action of short or long wave lengths when the quantities are measured by an open ionization chamber. He was comparing superficial roentgen rays of two different wave lengths (0.2 and 0.17Å) obtained with 30 kv. and 100 kv., roentgen rays in the neighborhood of those used with our "contact" apparatus.

For deep roentgen therapy, if the Coutard or Pfahler methods seem more efficient than the others, it is probably by virtue of the fact that more radiation is given in a shorter time. Contact therapy lends itself to destruction of superficial malignancy without much damage to surrounding healthy tissues; therefore, single massive dose techniques may be developed more easily than is possible with other forms of roentgen therapy.

SUMMARY

Determinations were made of the size of a single massive dose of roentgen rays with the Chaoul contact therapy apparatus necessary to effect destruction of rat sarcoma 39 without causing death of the animal. It was found that a dose of 10,000 r given to the tumor when it was 10 to 15 mm. in diameter, usually fifteen to nineteen days after transplant, effected destruction of the tumor with survival of the animal. When varying periods of time ranging from seven to seventy days were taken to deliver this same total dose (10,000 r), the longer the period, the more diminished the effect; in other words, the shorter the intervals between divided doses in which a given selected total dose of radiation is given, the greater the biologic effect. Contact therapy lends itself to the application of large tumor destructive doses with much less relative perifocal normal tissue damage than is encountered with other forms of roentgen irradiation; single or relatively few divided doses in short intervals of time may be given with greater safety to produce the optimum biologic effect.

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THE DEPENDENCE OF THE BIOLOGICAL EFFECT OF RADIATION ON INTENSITY AND WAVE LENGTH

AS MEASURED BY THE DELAYED LETHAL ACTION ON CHICK EMBRYOS

By D. E. LEA
Strangeways Research Laboratory
CAMBRIDGE, ENGLAND

I. INTRODUCTION

THE variation of the effect of radiation with alteration of wave length and intensity has been studied by a number of workers on various biological materials. In the case of the simplest actions of radiations, namely the inactivation of viruses and the production of gene mutations in, for example, *Drosophila*, the relationship of the biological effect to wave length and intensity is both known and understood (Timoféeff-Ressovsky;[15] Zimmer and Timoféeff-Ressovsky;[17] Lea;[8] Lea and Smith;[10] Gowen[3]). In these reactions the effect of a given dose of radiation is independent of intensity, and densely ionizing radiations (soft roentgen rays, neutrons or alpha particles) are somewhat less effective per roentgen than less densely ionizing radiations (gamma rays or hard roentgen rays). Certain other actions of radiation, in particular the lethal action on bacteria (probably to be interpreted as the production of lethal mutations) behave similarly to intensity and wave length variation, and likewise find a ready explanation (Lea and Haines[9]).

These are the simplest actions of radiation, in which the biological effect is due to the direct action of a single ionization on a particular structure of the organism. It is not certain whether there are any actions of radiation, other than those listed above, and perhaps also the production of chromosome abnormalities, which are of this simple type. It is at any rate certain that there are many reactions in which the effect cannot be interpreted as a specific action of a single ionization on a particular structure. In these cases it is likely that the radiation acts by the intermediary of chemical change, very possibly not directly affecting the structure which is eventually influenced but producing some active chemical in the cell which then acts biologically in much the same way as it would if it could be introduced into the cell by some other method.

The general rule in this class of reaction (if one is rash enough to generalize at the present stage) is for radiation at low intensity to be less effective than radiation at high intensity, and for the effectiveness of different wave lengths to differ, if at all, in the opposite sense to that described above. The variation with wave length is not at present understood, but the variation with intensity has received a quantitative explanation (Lea[7]), which is further discussed in the present paper.

Probably the simplest action of radiation which can be interpreted along these lines is the inhibition of cell division which radiation produces in various biological materials. Experiments on inhibition of division in irradiated tissue cells grown *in vitro* have given two arguments for interpreting the effects as due to a building up of some chemical change such as we have postulated. In the first place the effect of a given dose is found to vary with the intensity, being small at low intensities and increasing to a more or less steady value at high intensities (Canti and Spear;[2] Spear and Grimmett[14]). Now this type of variation suggests some sort of recovery process, and it has been found possible to explain the phenomenon quantitatively (Lea[7]) on the

assumption that the radiation produces a concentration of some chemical in proportion to the dose administered, and that this substance is removed by the cell's metabolism, so that after the removal of the radiation the concentration falls off exponentially.

There are, of course, other ways of explaining a recovery process, but the validity of the above interpretation is strengthened by the second argument, which is due to Spear, Gray and Read.[13] These authors pointed out that the ionization by neutrons and gamma rays is approximately (i.e. within a factor of two or three) equally effective, although the ionizations produced by the gamma rays are distributed almost uniformly throughout the cells while ionization produced by neutrons in a cell is localized along a small number of tracks with comparatively large intervals (of the order of a micron) between them completely without ionization. The fact that the effects per ionization are of the same order of magnitude in these two cases suggests strongly that the effect of radiation is not a direct one upon specific structures which are affected by ionization, but indirectly via the intermediary of chemical change in some constituent widely distributed through the cell.

The plausibility of this idea is increased by the similarity of the radiation phenomena to the phenomena of inhibition of division following mechanical injection of a suitable chemical into an organism. Colchicine is a chemical having a specific action on cells in mitosis, its effect being to hold them up in metaphase. The quantity required to be injected into a chick embryo to produce appreciable delay is equivalent to a concentration in the tissue of the order of 10^{-7} gm. per cm.[3] This is of the same order of concentration as would be expected of whatever chemical substance is produced by the radiation (assuming one molecule formed per ionization). Moreover, the delay lasts only for a limited period, during which the organism is excreting the colchicine. Some preliminary experiments by Hughes and Fell in this laboratory have given results which suggest that the holding up of division by colchicine may be interpreted along similar lines to the cumulative dose theory of the action of radiation. A certain threshold concentration of colchicine is required to hold up division. When a larger dose is administered the delay lasts until the organism's metabolism has reduced the concentration below this threshold value. In this way the dependence of the duration of the delay on the dose administered may be worked out, just as in the radiation theory.

In the present paper work which has been done in this laboratory on the effect of intensity and wave length variation on tissue cells grown *in vitro* (Canti and Spear;[2] Spear and Grimmett;[14] Lasnitzki and Lea[6]) is followed up by experiments on chick embryos irradiated *in ovo*. The results are surprisingly comparable, though the criterion of effect is entirely different. The experiment consisted simply in irradiating the embryos at the desired wave length and intensity and examining them two or three days later to see whether they were alive or dead.

Experiments on the action of radiations on chick embryos have been made by other authors (Wilson,[16] and references there cited), whose accounts should be consulted for a description of the changes occurring in the embryo and the area vasculosa after irradiation. Here we are content to use death as an easily recognizable criterion of effect for the purpose of comparing the relative efficiencies of high and low intensities, long and short wave lengths. The simplicity of some of the results obtained (see particularly the account of the dependence of effect on intensity, section 3) from an experiment on a highly complex organism is probably due to death being caused by damage to a group of cells which (as regards behavior to radiations) are all of the one type, and not the combined result of damage to a variety of cell types having different radiosensitivities and recovery periods.

The experiments described in this paper were made to investigate two phenomena: (*a*) the variation with intensity of the dose of roentgen rays required to produce a given biological effect, and (*b*) the relation between the doses of roentgen and gamma rays needed to produce the same biological effect. The first investigation is complete in itself; the second necessarily depends upon it. This is so because the effect of a given dose is not independent of the intensity unless the intensity is rather high, higher than can be given to an object as large as a hen's egg with the sources of radium available. In comparing the efficiencies of the two radiations, therefore, it is necessary first to understand the effect of intensity variation.

2. EXPERIMENTAL METHODS

The radiations used were roentgen and gamma rays. The roentgen rays were generated at approximately 160 kv. constant potential and filtered by 0.7 mm. Cu + 1.2 mm. Al. The half-value thickness of copper was 1.0 mm., corresponding to an effective wave length of 0.15 Å. The eggs to be irradiated were placed in an incubator underneath the tube at various distances depending on the intensity required, and the intensities were measured by a graphite ionization chamber placed at various points in the incubator tray. The intensity in the tray varied over an extreme range of ± 7 per cent from the mean intensity. Intensities measured with the graphite chamber were converted to roentgens by calibrating the chamber against a free air ionization chamber. At the highest intensity used only a single egg could be irradiated at a time. At the lower intensities as many as 30 were irradiated together and batches removed at intervals.

The gamma irradiations were made, 1 egg at a time, in an incubator. The radium available consisted of 760 mg. in four units, these being arranged to give the greatest uniformity of intensity over the embryo consistent with the intensity not being too low. The intensity was measured by a graphite ionization chamber, it having been shown (Gray[4]) that such chambers measure gamma-ray intensities in roentgens to about 1 per cent.

The eggs, which were incubated for six days prior to irradiation, were opened two or three days after irradiation and the embryo recorded as alive or dead. The choice of time elapsing between irradiation and opening needed some consideration. A very large dose would be required to produce immediate death, since death is the conclusion of a chain of reactions started by the irradiation, which takes some time for its completion. The dose required to produce death at a time, T, after irradiation diminishes at first rather rapidly with increasing T, and the lethal dose deduced would depend rather markedly on the value of T chosen. This makes the comparison of different intensities difficult, since when the exposure to high intensity takes, say, five minutes, and to low intensity five hours, it is likely to make a difference whether T is measured from the beginning, middle or end of the exposure. For this reason a time as short as twenty-four hours elapsing between exposure and opening was found unsuitable, and a period of either two or three days was used, it being found that there was very little difference between the results obtained at these two times.

3. VARIATION OF EFFECT WITH INTENSITY (ROENTGEN RAYS)

Four intensities were used, and several different times of exposure at each intensity. At each exposure time from 4 to 15 eggs were treated, and the proportion of embryos surviving was plotted against the dose. If on opening a batch of N eggs A are found to be alive and D dead, the surviving fraction is $A/N \pm (A \cdot D/N^3)^{\frac{1}{2}}$ since A is distributed according to the binomial frequency distribution. The error indicated is the standard deviation, and is represented by the vertical lines in the graphs (Figs. 1, 3 and 4). The odds are 2:1 against the value which would be obtained for the surviving fraction if a very large number of

eggs were irradiated lying outside the limits drawn in this way. When either A or D is zero the formula $A/N \pm 1/(N+1)$ is used instead.

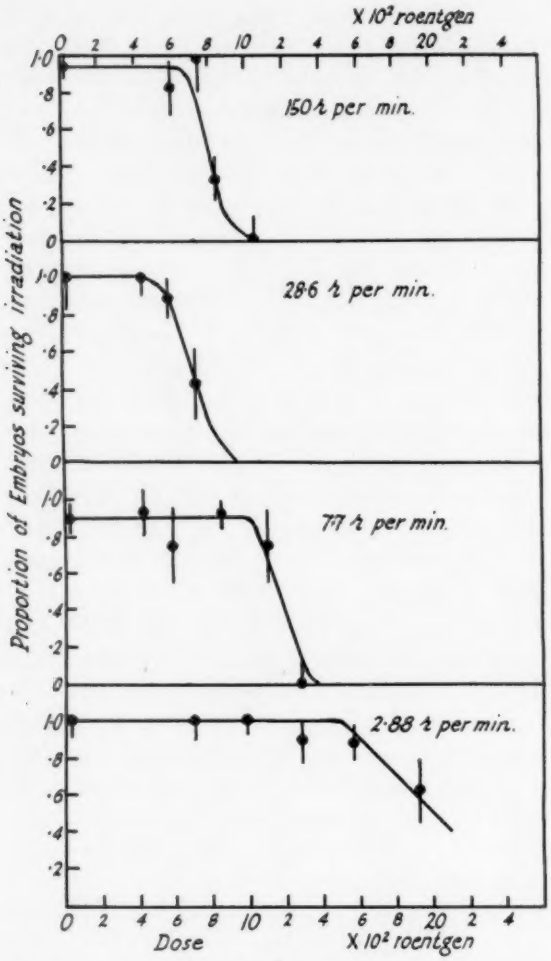

FIG. 1. Lethal effect of various intensities of roentgen rays (0.15Å) on chick embryos.

These values are plotted against the doses in Figure 1, and from these survival curves the doses required for 50 per cent survival are deduced. This is taken to be the lethal dose for an average embryo; the fact that some embryos survive larger doses and some are killed by smaller doses is of course due to biological variation. It is evident that the lethal dose is fairly constant at high intensities, and increases at low intensities. Figure 2 shows the lethal dose deduced in this way plotted against the intensity. This variation with intensity is of the type to be expected when a recovery mechanism is present by which the organism is able to counteract the effect of the radiation. If the radiation is administered slowly the recovery mechanism almost keeps pace with the damaging effect of the radiation, and a very large dose is required to kill the embryo. If, on the other hand, the radiation is administered quickly, i.e. at high intensity, the recovery mechanism is quite unable to cope with the damage done by the radiation, so that the lethal dose is low and is practically unchanged by further increase of intensity.

If simple assumptions are made as to the mechanism of the recovery process, the form of the curve of lethal dose against intensity can be predicted quantitatively. In an earlier paper (Lea[7]) it was shown that a number of different assumptions of the mechanism gave much the same curve, and this curve agreed with such experimental data as were available to test it. We shall here confine ourselves to one of the suggested recovery mechanisms which seems the most plausible.

We suppose, in accordance with the discussion in the introduction, that the eventual lethal effect follows damage by

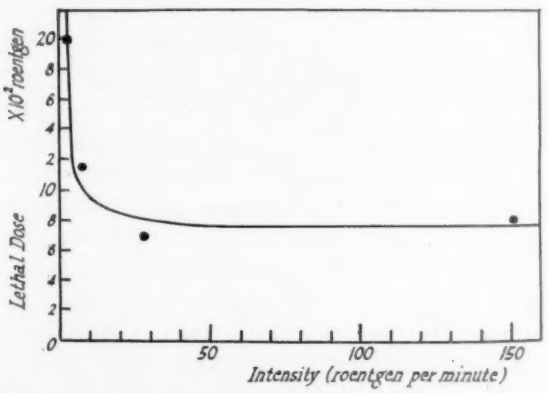

FIG. 2. Lethal dose of roentgen rays (0.15Å) of various intensities (curve theoretical, points experimental).

the radiation to a certain group of cells, and that the radiation effects this damage by causing the appearance of some harmful chemical in the cell. The cellular metabo-

lism removes this chemical at a rate proportional to its concentration. A short irradiation at high intensity would produce a concentration proportional to the dose administered, but when the radiation is administered slowly or in a series of fractions with intervals between, the concentration at the end of the treatment is less than if the whole dose were given in one short high intensity exposure.

To avoid elaborating the hypothesis unnecessarily, we prefer to speak not of the concentration of chemical produced in the cell, but of the "cumulative dose" (cf. Hoffmann and Reinhard;[5] Quimby and MacComb[11]) existing at any moment. Immediately after a short irradiation at high intensity the cumulative dose is just equal to the dose administered. With lapse of time it diminishes. After irradiation which has been spread over a prolonged time either by fractionation or by the use of low intensity, the cumulative dose is less than the total dose administered. We suppose that in the present experiments death ensues if the cumulative dose rises above a certain threshold (D_1). Along these lines (cf. Lea[7]) we may calculate the rate of variation of lethal dose with intensity. At a given moment the rate of increase of the cumulative dose is equal to the intensity I, the rate of decrease is proportional to the cumulative dose existing at that moment and may be written D/τ, where τ may be described as the characteristic recovery time of the material. Thus the net rate of increase is given by

$$\frac{dD}{dt} = I - D/\tau. \qquad (1)$$

Integrating this equation we obtain the time (t) needed at constant intensity I to obtain a cumulative dose D_1 at the end of the exposure. It is given by the equation

$$It = I\tau \log_e \frac{1}{1 - D_1/I\tau}. \qquad (2)$$

This equation shows that at sufficiently high intensity the actual dose (It) needed is just equal to D_1, and that there is a critical intensity $I_0 = D_1/\tau$, below which no irradiation, however prolonged, will enable the cumulative dose to be attained. Writing

$$y = \frac{\text{actual dose required}}{\text{threshold dose}} = It/D_1$$

$$x = \frac{\text{critical intensity}}{\text{actual intensity}} = I_0/I = D_1/I\tau$$

we have

$$y = \frac{1}{x} \log_e \left(\frac{1}{1-x} \right). \qquad (3)$$

Evidently to fit the experimental points shown in Figure 2 we must take D_1, the lethal dose at very high intensity, to be about 750 roentgens. Using this value, and taking $\tau = 280$ minutes, which is found by trial to give the best fit, we deduce the theoretical curve given in Figure 2, which is seen to represent the experimental results quite well.

The critical intensity below which, according to the theory, death should not occur with irradiation, however prolonged, is $750/280 = 2.68$ r per minute. It should not, however, be imagined that if embryos were irradiated at an intensity of, say, 2.5 r per minute it would be impossible in practice to kill them by sufficiently prolonged irradiation. To begin with, different cells of the group we are considering doubtless show a certain amount of variation in their rate of recovery, the effect of which will be to make the upward bend of the curve of Figure 2 more gradual. Also, there are doubtless other groups of cells in the embryo which, with sufficiently large doses of radiation, could be damaged sufficiently to lead to the death of the embryo, and in which the recovery is slower. A calculated curve based on our simple theory, therefore, especially with complicated biological materials, must not be expected to hold down to very low intensities. The fact that it fits over the range of the present experiments tends to confirm that the eventual lethal effect is due to the action of radiation on a single type of cell, and that there is no

second group of cells which can be sufficiently damaged to contribute markedly to the death of the embryo by doses up to 2,000 roentgens.

4. Fractionation of Dosage (Roentgen Rays)

On our theory, division into a number of fractions should diminish the effect of a given dose in much the same way as its administration at low intensity, since there is (on the theory) hardly any difference between the cumulative dose existing at the end of a period during which irradiation has been continuous at low intensity, and the cumulative dose existing at the end of a similar period during which the radiation has been administered in a number of equal and equally spaced fractions, even though the separate fractions were given at high intensity. This conclusion may of course not be valid in a complicated case in which more than one group of cells is concerned, but we should expect it to hold with the chick embryos since the indications are (cf. previous section) that here the lethal effect is mainly due to damage to a single type of cell.

Ancel and Vintemberger[1] have reported experiments with chick embryos in which the effect of a given dose was found to be reduced by spreading it over seven hours in a series of fractions.

The present experiments were made (for reasons outside my control) with roentgen rays of a different wave length from the series described in the previous section. It is unlikely that the recovery process will be different on account of this wave length difference. The radiation was generated at approximately 60 kv. constant potential with a filtration of 2.4 mm. Al. The half-value thickness was 3 mm. Al corresponding to an effective wave length of 0.363 Å. The eggs were irradiated singly, the intensity at the embryo being 39.7 r per minute. In each experiment the eggs were divided into two batches. The eggs of one batch were irradiated several times for periods of four minutes, at intervals of from sixty to ninety minutes. The eggs of the other batch received the radiation concentrated in a single exposure at the same intensity or in two or three exposures separated by short intervals.

Table I

Comparison of Fractionated and Concentrated Doses

(Soft roentgen rays, 0.363 Å, 39.7 r per min.)

Concentrated Dose

Duration of Exposure	Number of Embryos		Proportion Surviving
	Alive	Dead	
0 min.	15	5	0.75 ± 0.10
10	6	2	0.75 ± 0.16
12	4	1	0.80 ± 0.19
16	6	2	0.75 ± 0.16
18	2	5	0.29 ± 0.17
20	1	3	0.25 ± 0.22
27	0	7	0.00 ± 0.12

Fractionated Dose

Total Exposure	How Administered	Alive	Dead	Proportion Surviving
24 min.	6 × 4 min.; 76 min. interval	6	1	0.86 ± 0.13
40 min.	10 × 4 min.; 85 min. interval	4	3	0.57 ± 0.19

The results obtained are given in Table 1. In Figure 3 the survival curve for the concentrated irradiations is shown, from which it is deduced that a survival of 0.57 ± 0.19 corresponds to a concentrated irradiation of 17 ± 2 minutes. Thus a total exposure of forty minutes divided into ten fractions at eighty-five minute intervals only produces the same effect as a concentrated dose of seventeen minutes. This observation is, of course, to be expected on the cumulative dose theory, and is interpreted to mean that the cumulative dose at the end of the fractionated treatment is equal to that delivered in seventeen minutes' concentrated dose.

By applying the theory to these observa-

tions, we may deduce the characteristic recovery time τ which enters into the theory by the following procedure: The first four minutes' exposure is given at a time 9×85 minutes before the end of the treatment, and its contribution to the cumulative dose at the end of the treatment is thus $4\,e^{-9\times85/\tau}$. Similarly for the second fraction we obtain $4\,e^{-8\times85/\tau}$, and so on. The cumulative dose at the end of the exposure, due to the whole ten fractions, thus amounts to

$$4\{x^9+x^8+x^7+\cdots x^0\}=4\cdot\frac{1-x^{10}}{1-x} \qquad (4)$$

where $x=e^{-85/\tau}$.

Equating this to 17 ± 2 we deduce that $\tau=350\pm46$. A small correction is needed for the fact that the seventeen minute concentrated dose was not actually delivered in negligible time, and when this correction is made we find

$$\tau=320\pm46 \text{ minutes.}$$

This value is seen to agree well with the value of 280 minutes which was found by

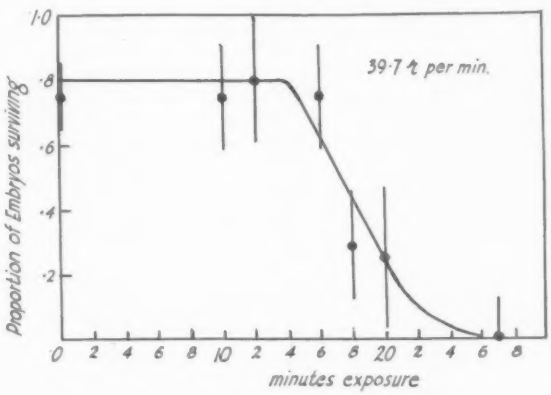

Fig. 3. Lethal effect of soft roentgen rays (0.363Å) on chick embryos.

trial to give the best agreement between the experiments of the previous section and the cumulative dose theory. It is evident, therefore, that with this material the effect of fractionation, at any rate with the intervals and dose used, is not in principle different from the effect of diminishing the intensity and that both are describable in terms of the same theory of recovery.

5. Variation of Effect with Wave Length

In the course of these experiments two comparisons of the effectiveness of different wave lengths were made. A comparison of medium (0.15 Å) and soft (0.363 Å)

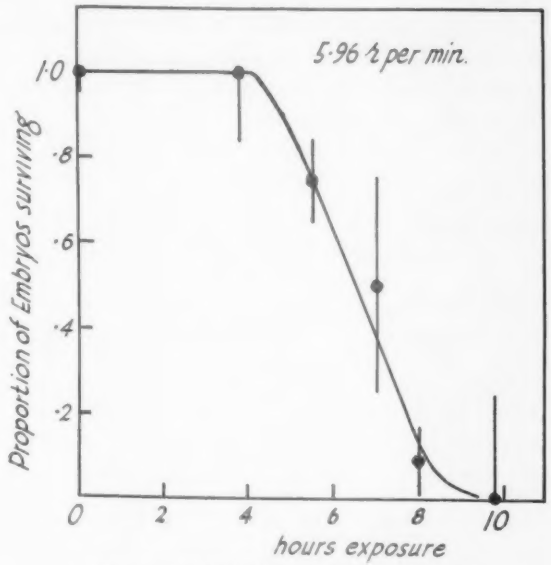

Fig. 4. Lethal effect of gamma rays on chick embryos.

roentgen rays is provided by Figures 1 and 3. The intensity (39.7 r per min.) used in the soft roentgen-ray experiment summarized in Figure 3 was between the two highest used in the medium roentgen-ray experiments, and in this region the lethal dose is independent of the intensity, and may be read off from Figure 2 to be 760 roentgens for medium roentgen rays. The exposure time for 50 per cent survival of embryos treated with soft roentgen rays at 39.7 r per minute is seen from Figure 3 to be 17.5 minutes, so that the lethal dose is $17.5\times39.7=695$ r. Thus 695 r of soft roentgen rays and 760 r of medium roentgen rays are equally effective.

The comparison between medium roentgen rays and gamma rays was more difficult because in the region of intensity in which it is necessary to work with the gamma-ray source available the dose varies rapidly with intensity. The survival curve with gamma rays at 5.96 r per minute (Fig.

4) indicates that an exposure of 6.5 hours kills half the embryos. By graphical interpolation between the data of Figure 1 we can deduce what intensity of medium roentgen rays would also kill half the embryos in 6.5 hours; this intensity is 3.55 r per minute. It is evident that when delivered in equal times 1 r of gamma rays produces the same effect as $3.55/5.9 = 60.60$ r of medium roentgen rays. We thus deduce the data of Table II.

Table II

DOSES OF VARIOUS WAVE LENGTHS BIOLOGICALLY EQUIVALENT TO 1 ROENTGEN OF GAMMA RAYS

Radiation	Wave Length	Dose
Gamma rays	0.014 A	1.0
Medium roentgen rays	0.15 A	0.60
Soft roentgen rays	0.363 A	0.55

This variation of biological effect with wave length is similar to that found for inhibition of division in tissue cultures (Lasnitzki and Lea[6]). Moreover, the characteristic recovery time found in the present experiments ($\tau \sim 300$ minutes) is not very different from that found in the analysis of the effect of variation of intensity in the tissue culture experiments ($\tau \sim 217$ minutes).

I am greatly indebted to Dr. F. G. Spear, Dr. A. F. W. Hughes and Dr. A. Glücksmann for cooperation in these experiments, and to the British Empire Cancer Campaign for its support; also to the Medical Research Council for the loan to the Strangeways Laboratory of some of the radium employed.

SUMMARY

Experiments are described in which the dependence upon intensity and wave length of the dose required to produce a given biological effect was studied. Chick embryos irradiated after six days' incubation were used, and the criterion of effect was death two to three days after irradiation. When a given dose is delivered in a single exposure at intensity greater than about 10 r per minute the effect is practically independent of intensity. The effect is reduced, however, by spreading the dose over a long period, either by continuous irradiation at low intensity, or by division into a series of high intensity fractions with intervals between. These results are explained on the idea that recovery from the effects of radiation can occur, and that when the radiation is administered at low intensity or with suitable intervals of rest, the recovery processes practically keep pace with the damage produced by the radiation. Good agreement between the experiments and the theory is obtained when the theory is made quantitative by the use of the conception of "cumulative dose." The doses of gamma rays (0.014 Å) and of medium (0.15 Å) and soft (0.363 Å) roentgen rays required to produce the same effects are compared and are found to be in the ratio 1.0:0.60:0.55.

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THE PRODUCTION AND MEASUREMENT OF SOFT ROENTGEN RAYS FOR BIOLOGICAL EXPERIMENTS

By D. E. LEA
The Strangeways Research Laboratory
CAMBRIDGE, ENGLAND

I. DESIGN OF THE ROENTGEN TUBE

THERE are a number of purposes for which the use of roentgen rays softer than those employed in therapy has advantages in research on the biological action of radiations. This is particularly true in experiments on the action of radiations on bacteria and viruses, where the dependence of the effect upon the wave length of the radiation yields important information on the mechanism of the action of the radiations, and where the most significant variation with wave length occurs in the region of wave lengths longer than 1 Å. These materials are in general highly resistant, and high intensities of radiation are necessary.

It is a fortunate circumstance that with a suitable design of roentgen tube an output of radiation which, expressed in roentgens, is very high can be obtained with a low power input. Further, by the use of appropriate materials for the target of the tube one can excite the characteristic K or L spectrum of the target material, and by the use of suitable filters this characteristic radiation may be obtained almost free from any background of continuous spectrum. High intensities of almost monochromatic roentgen rays are thus obtained much more readily in the long wave region of 1 to 10 Å than in the region of shorter waves.

A number of authors have described tubes for the production of wave lengths of 1 to 10 Å. Some of these are of the hot cathode type (Holweck, Dauvillier, Thoraeus, Södermann), others of the ionic type (Kersten, Band). For the softer radiations, the ionic type has an advantage since when a hot filament is employed special measures are necessary to avoid the deposition of tungsten on the target. The output from an ionic tube, however, does not increase indefinitely with increasing tube current when operating at very low voltages, and this sets a limit to the output from this type of tube which does not apply to the hot cathode type. However, I have found it possible to obtain a very high intensity from an ionic tube exceptionally simple in design and economical in running, by making the window and the target one and the same metal foil. By this means it is possible to bring the specimen to be irradiated very close to the target. At a few millimeters from the target the intensity falls off rapidly with increasing distance, but since specimens irradiated with soft radiations are necessarily very thin, this is no disadvantage.

The tube is shown diagrammatically in Figure 1. A single water cooling jacket suffices. The cathode is of aluminium, and the target is a foil which is soldered or waxed over an aperture in a brass disc which fits into a depression in the end of the tube. The insulator is a pyrex cylinder, all joints being made with apiezon putty. The brass discs holding different targets may be interchanged in a minute or two. A two stage mercury vapor pump (without vapor trap) backed by a rotary oil pump evacuates the tube and the pressure is adjusted by a commercial needle-valve leak.

The body of the tube is at earth potential, and the cathode is supplied at a negative potential of 3 to 40 kv., depending on the radiation which is being excited. For the supply of potentials up to 10 kv. which are adequate for exciting wave lengths longer than 4 Å, an inexpensive full wave rectifier set was made from a neon sign transformer, a pair of small rectifying valves, and two condensers. For higher potentials it was found convenient to use

the negative half of a Greinacher set designed to operate a larger tube. A resistance of several hundred thousand ohms is necessary between the generator and the tube, since an ionic tube has a negative slope resistance. The current through the tube can be increased by using a number of small targets in place of one large one, i.e. by using as a target a foil stretched over a grid of brass, and this was done when the largest intensities were required. The maximum heat dissipated by the target being

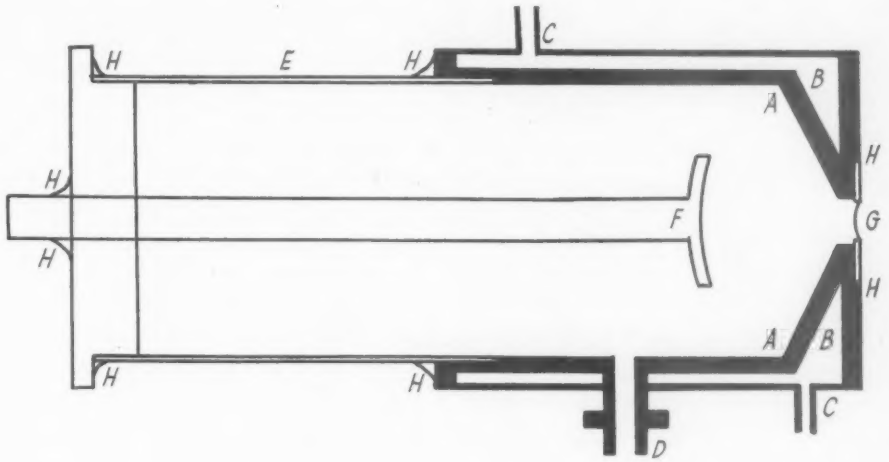

FIG. 1. Roentgen-ray tube (vertical section). *A*, body of tube; *B*, water jacket; *C*, water ports; *D*, vacuum port; *E*, pyrex glass cylinder; *F*, aluminium cathode; *G*, window-cum-target; *H*, vacuum seals (apiezon putty). The diameter of the tube is about 8 cm.

is regulated by means of the needle valve leak, and remains fairly constant once the tube has settled down to steady running conditions, which may take an hour if the tube has been out of use for some time, otherwise only a few minutes. A device may be built to make automatic adjustment to the needle-valve to enable the tube to run for long periods without attention (Lea[5]).

The radiation is transmitted through the target and is too soft to permit of the use of cooling water behind the target. The heat generated in the target is therefore dissipated principally by conduction to its edges. If heat H is generated uniformly over a circular target of thickness d and thermal conductivity k, then the highest temperature will be at the center and will be T above the temperature at the rim, where $T=H/4\pi kd$. If this rises above a certain value the target will puncture. Since T is independent of the radius of the target, the maximum dissipation of heat proportional to d, and the proportion of radiation transmitted (normally) being $e^{-\mu d}$, the greatest intensity is produced when the target thickness is chosen to make $\mu d=1$, where μ is the absorption coefficient. Actually, since it is desirable to obtain nearly monochromatic radiation, which can best be done by filtering through a further thickness of target material, values of μd as high as 3 are sometimes used. The tube is usually operated at a voltage about three times the voltage of the radiation it is desired to excite, and the maximum input is 1 to 2 ma. The aluminium target is 20μ thick and the maximum intensity obtained (for a target supported on a grid) is 8,000 roentgens per second. The copper target is 66μ thick, and the maximum intensity obtained is about 1,500 r per second. The silver foil of thickness 6μ used when silver L-radiation is required is not sufficiently strong and hole-free to hold the vacuum, and the window is therefore made double, the inner foil

being silver and the outer foil cellophane 30μ thick. The maximum intensity under these conditions is about 800 r per second.

2. METHOD OF DOSAGE-RATE MEASUREMENT

The measurement of the dosage-rate received by the irradiated specimen presents some problems. At the position occupied by the specimen (1 to 5 mm. from the tube window) the radiation is highly divergent and its intensity is diminishing rapidly with distance from the window. The specimen itself is usually less than 10μ thick, so that the roentgen-ray intensity is uniform throughout it. It is not possible, however, to make an ionization chamber so thin; all that can be done is to make the window of the chamber coincide with the position occupied by the specimen. Some means is therefore required for measuring in an ionization chamber of finite depth the dosage-rate at its front.

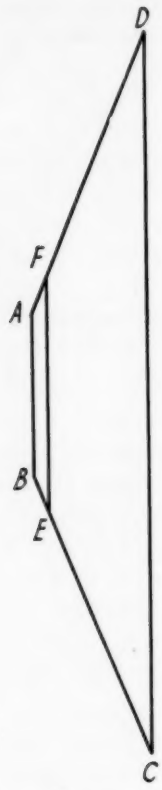

Fig. 2

The method which has been adopted is to make the ionization chamber in the form of the frustrum of a cone. The narrow end is closed by a thin window through which the radiation enters, and which is brought into coincidence with the plane normally occupied by the specimen. Figure 2 represents a section of the chamber. The window AB is of thin aluminium foil or of cellophane and has a diameter of 0.45 cm., approximately equal to the diameter of the specimens usually irradiated. The collecting plate CD is a disc of aluminium 2.0 cm. in diameter. The depth of the chamber (d) is 0.319 cm. Radiation entering the chamber at an angle as great as 70° to the axis traverses the full depth of the chamber. The collecting plate is insulated by amber from an earthed guard ring. $ABEF$ represents the ideal very shallow chamber of depth δ which would measure the dosage-rate at the plane AB. A quantum of radiation entering the chamber at an angle θ with the axis ($\theta \leqq 70°$) will have probability $\mu d \sec\theta$ of being absorbed in the volume $ABCD$ and probability $\mu\delta \sec\theta$ of being absorbed in $ABEF$, providing that both probabilities are small. The ionizations produced in the volumes $ABCD$, $ABEF$ are thus in the ratio d/δ. If the measured ionization current is i, the ionization per unit volume in a very thin layer near the window AB, supposed of area A, is thus i/Ad. If the absorption in the air of the chamber is appreciable, this formula becomes modified to

$$\frac{\mu d \sec\theta}{1-e^{-\mu d \sec\theta}} \cdot \frac{i}{Ad}.$$

An average value of the angle θ was found by measuring the absorption produced by thin aluminium foils placed between the tube and the ionization chamber when the latter was in its standard position, and comparing with the absorption for normal incidence. The effective value of sec θ was found to be 1.19. The same experiment enables a correction to be made for the absorption of the radiation

by the window of the chamber in ionization measurements.

As a general check on the method, extra spacing rings were made for the ionization chamber, and measurements were made, with three different depths of chamber, of the ionization produced by a given intensity of aluminium K-radiation. The results are shown in Table 1.

TABLE I

Chamber Depth (cm.)	Ionization Current (e.s.u. per sec.)	Ionization Per Unit Volume (relative)
0.177	0.502	1.026
0.319	0.800	1.000
0.507	1.081	0.956

The third column shows the ionization dosage-rate at the window relative to the dosage-rate with the standard chamber depth of 0.319 cm., correction being made for the absorption in air as described above. The variation in the last column is probably to be ascribed to the existence of a small proportion of radiation entering the chamber in directions inclined at large angles to the axis, and striking the walls before traversing the full depth of the chamber. Measurements are usually made with a chamber depth of 0.319 cm., and to correct for this very oblique radiation the ionization per unit volume at the window calculated from the observation is multiplied by 1.048, which is the figure obtained by extrapolating to zero chamber depth the figures in the third column of the table.

So far no account has been taken of ionization produced in the chamber by photoelectrons liberated in the walls. This is justifiable for aluminium K-radiation, since the range of the photoelectrons is very small compared with the chamber dimensions. In the case of copper K-radiation, while the greater part of the ionization is to be ascribed to photoelectrons liberated in the air, there is a small proportion due to photoelectrons liberated from the walls. This produces an appreciable error if the walls are of aluminium, but the error is inappreciable if the chamber window is of cellophane and the collecting plate and walls are graphited.

It is of course essential that the window of the chamber shall, during measurements, always be brought to exactly the same distance from the target of the tube as that at which the specimen has been irradiated. To facilitate this a pair of rails are provided along which a a carriage carrying the ionization chamber can slide, bringing the chamber to a fixed and reproducible distance from the window of the tube. A second carriage interchangeable with the first carries the specimen to be irradiated, which can by this means be brought accurately to the position formerly occupied by the window of the ionization chamber.

The ionization currents are measured either by passing them through a resistance of 10 megohms and measuring the potential developed with a quadrant electrometer, or by a valve electrometer instrument, the former method being more convenient for currents greater than 10^{-9} amp and the latter for smaller currents. The measurements of the dosage-rate at the specimen are probably correct to about 5 per cent.

3. THE HOMOGENEITY OF THE RADIATIONS

In an investigation of the variation of biological effect with wave length it is of course desirable to work with narrow regions of the spectrum, though the degree of homogeneity obtainable by a spectrometer is of course unnecessary. A satisfactory degree of homogeneity can be obtained by running the tube at a voltage two or three times as great as the voltage of the characteristic radiation it is desired to excite, and filtering the radiation by a thickness of anticathode material sufficient to reduce the intensity of the characteristic radiation to about one-tenth of its original intensity. An estimate of the degree of homogeneity obtained can be made by plotting the absorption curve of the radia-

tion in some material not having an absorption edge in the region under investigation.

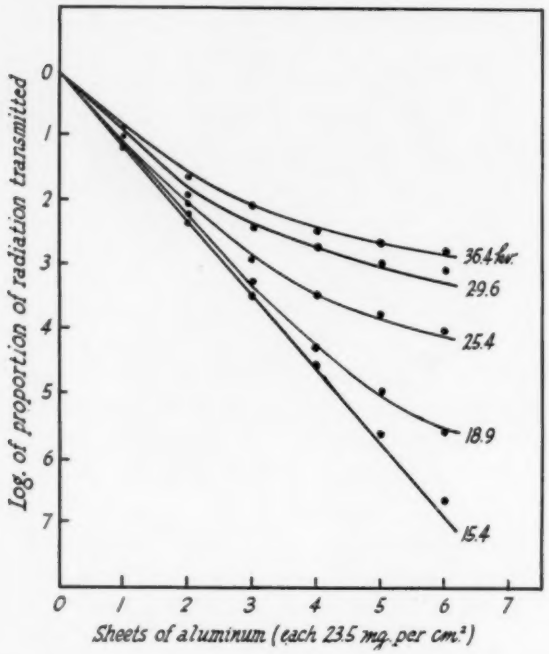

FIG. 3. The absorption of copper radiation in aluminium.

As an example, Figure 3 shows the absorption curves in aluminium obtained when the tube is provided with a target of copper of thickness 59 mg/cm.². At the lowest voltage (15.4 kv.) the radiation is evidently very nearly homogeneous and has an absorption coefficient of 49.7 gm^{-1} $cm.^2$ This is the copper K-radiation, of quantum energy 8 kv. It is safe to assume that at other tube voltages no appreciable quantity of radiation exists below 7 kv. or between 8 and 15 kv., since 98 per cent of such radiation would be absorbed in the anticathode on account of the absorption edge phenomenon. As a rough basis of calculation one can assume that the radiation at higher tube voltages consists of the copper K-radiation having an absorption coefficient of 49.7 gm^{-1} $cm.^2$, and a harder component of about 15 kv. having an absorption coefficient of 8.5 gm^{-1} $cm.^2$, in aluminium. On this basis compositions can be found by trial for the radiation at various tube voltages which fit the observed absorption curves. The curves of Figure 3 are calculated in this manner, assuming the following compositions for the radiation at various tube voltages:

Tube kv.	15.4	18.9	25.4	29.6	36.4
Copper radiation	100%	99%	95%	88%	81%
Hard radiation	0%	1%	5%	12%	19%

The percentages are given in terms of the ionization in small chambers, not in terms of numbers of quanta.

A similar analysis for aluminium K-radiation when the tube was equipped with an aluminium target of 5.44 mg/cm.², and a further filter of 2.37 mg/cm.² was present in the shape of the chamber window, was made by measuring the absorption in cellophane. The experimental absorption curves were analyzed into two components of which the softer was the aluminium K-radiation and the harder was assumed to be of 2.6 kv. quantum energy. The following percentage compositions fitted the experimental absorption curves:

Tube kv.	2.5	3.0	3.5	4.0	4.5	5.0
Aluminium radiation	100.0%	99.99%	99.9%	99.3%	98.6%	97.3%
Hard radiation	0.0%	0.01%	0.1%	0.7%	1.4%	2.7%

This table illustrates the very high degree of homogeneity which can be obtained in the long wave region by making use of the absorption edge phenomenon.

My thanks are due to Mr. Hignell for his skillful construction of the roentgen tube and to the British Empire Cancer Campaign for a grant to the Strangeways Laboratory defraying the cost of the roentgen-ray apparatus.

SUMMARY

An account is given of a simple design of continuously evacuated roentgen tube operating at a voltage of 3 to 40 kv. and producing nearly homogeneous monochromatic characteristic roentgen rays of wave

lengths from 1.5 to 8.3 Å. In this tube one and the same metal foil serves as target and as window. By this means the specimen to be irradiated can be brought within a few millimeters of the target, and very high intensities, of the order of 1,000 roentgens per second, are obtainable, at very low power inputs. The method of measuring the dosage-rate, and the degree of homogeneity of the radiation, is also described.

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EDITORIAL

FRACTURES OF BOTH BONES OF THE FOREARM IN CHILDREN

FRACTURES of the shafts of both bones of the forearm in children is a fairly common injury, particularly so in the younger age group. Recently Bosworth[1] has reported the end-results in a series of cases which passed through the outpatient department and wards of St. Luke's Hospital, New York, during the seven year period from 1932 to 1938 inclusive. He made a critical analysis of all the shaft fractures of both bones of the forearm in children sixteen years of age and under and he reports a series of 50 cases. As would be expected, the males outnumbered the females two to one. The average age was nine years but almost two-thirds of the children were aged ten or under. As would also be expected, the greatest number of the fractures of both bones of the forearm were of the greenstick variety.

Bosworth found that the right arm was broken 24 times and the left 26. Most of the fractures resulted from a fall sustained while the children were playing. He did not include in his cases Colles' fractures and epiphyseal separations.

In the treatment of the fractures, for immobilization anteroposterior plaster splints were usually applied to include the wrist and palm but not the elbow. In a few of the cases in which there was overriding, the splint was made to include the elbow. Immobilization of the fractured arm was maintained for an average period of four weeks, and some form of physiotherapy was employed in about half of the cases.

In his follow-up of these 50 patients, Bosworth was able to trace 32. The follow-up studies included physical and roentgenologic examination as well as bilateral measurements of the forearm length and wrist circumference of each child and inspection of the carrying angle. The average follow-up period was five years. No case has been included which was seen less than one year and five months after initiation of therapy.

From an analysis of the results in his series of cases, Bosworth came to the same conclusion as did Whipple and St. John[2] in an analysis of their cases and Bagley[3] in an analysis of his cases, namely that open reduction of such fractures, that is fractures of both bones of the forearm, is unnecessary. Bosworth found that in no case did shortening of the forearm occur and in only one case was there a demonstrable decrease in the carrying angle, and it developed during the course of the study of this case that there was a refracture of the same arm which was treated in another hospital. In only 2 cases was there a difference in the circumference of the two wrists.

Bosworth's study of his cases, in the light of those of Whipple and St. John and of Bagley, serves to call attention to the excellent ultimate results ordinarily obtained by conservative treatment of this type of fracture in youngsters and that the failure to secure a 100 per cent anatomical re-position of the fragments is no indication for operative procedure. He emphasizes the point that it is surprising how often a good reduction can be obtained by simple manipulation, even in the presence of marked displacement of both bones. Happily, in contrast to adults, "children exhibit a phe-

[1] Bosworth, B. M. Fractures of both bones of the forearm in children. *Surg., Gynec. & Obst.*, March, 1941, *72*, 667–669.

[2] Whipple, A. O., and St. John, F. B. Cited by Bosworth.

[3] Bagley, C. H. Cited by Bosworth.

nomenal capacity to re-establish the normal length, alinement and continuity of fractured bones even when there is gross displacement and overriding after reduction and immobilization have been carried out."

Out of 25 patients treated by closed reduction who had had fractures of both bones of the forearm, in 21 both anatomical and functional results were excellent.

The roentgenographic results in this group of closed reductions were excellent in 22, good in 3 and fair in 3, and solid bony union was demonstrated in every case. What is of equal importance is that in no case did cross union occur between radius and ulna in these cases that were treated by closed reduction.

Bosworth concludes from this study that so satisfactory are the results of conservative treatment of these fractures in children that operative interference need rarely be employed, and in children with shaft fractures of both bones of the forearm complete anatomical reduction with end-to-end apposition of the fragments is not essential to excellent functional and cosmetic recovery. Bosworth does not imply by this statement that one should not make every effort to replace the fragments in as excellent position as is possible without open reduction but he does inveigh against needless open reduction in this type of fracture in children, with its potential dangers of infection.

It is well that the roentgenologist be familiar with the end-results that are obtained in this type of fracture where complete apposition is not obtained in order not to subject himself and the surgeon as well as the patient to a needless amount of radiation during an attempted roentgenoscopic reduction of fractures of both bones of the forearm in children.

SOCIETY PROCEEDINGS, CORRESPONDENCE AND NEWS ITEMS

Items for this section solicited promptly after the events to which they refer.

MEETINGS OF ROENTGEN SOCIETIES*

UNITED STATES OF AMERICA

AMERICAN ROENTGEN RAY SOCIETY
Secretary, Dr. C. B. Peirce, Royal Victoria Hospital, Montreal, Canada. Annual Meeting: Cincinnati, Ohio, Sept. 23–26, 1941.

AMERICAN COLLEGE OF RADIOLOGY
Secretary, Mac F. Cahal, 540 N. Michigan Ave., Chicago, Ill. Annual meeting: Hotel Statler, Cleveland, June 4, 1941.

SECTION ON RADIOLOGY, AMERICAN MEDICAL ASSOCIATION
Secretary, Dr. J. T. Murphy, 421 Michigan St., Toledo, Ohio. Annual meeting: Cleveland, Ohio, June 2–6, 1941.

RADIOLOGICAL SOCIETY OF NORTH AMERICA
Secretary, Dr. D. S. Childs, 607 Medical Arts Bldg., Syracuse, N. Y. Annual meeting, 1941: To be announced.

RADIOLOGICAL SECTION, BALTIMORE MEDICAL SOCIETY
Secretary, Dr. Walter L. Kilby, Baltimore. Meets third Tuesday each month, September to May.

SECTION ON RADIOLOGY, CALIFORNIA MEDICAL ASSOCIATION
Secretary, Dr. Wilbur Bailey, 2007 Wilshire Blvd., Los Angeles. Next annual session, Hotel Del Monte, Del Monte, May 5–8, 1941.

RADIOLOGICAL SECTION, CONNECTICUT MEDICAL SOCIETY
Secretary, Dr. Max Climan, 242 Trumbull St., Hartford, Conn. Meets bi-monthly on second Thursday, at place selected by Secretary. Annual meeting in May.

SECTION ON RADIOLOGY, ILLINOIS STATE MEDICAL SOCIETY
Secretary, Dr. H. W. Ackemann, 321 W. State St., Rockford, Ill.

RADIOLOGICAL SECTION, LOS ANGELES CO. MED. SOC.
Secretary, Dr. Wilbur Bailey, 2007 Wilshire Blvd., Los Angeles, Calif. Meets on second Wednesday of each month at County Society Building.

RADIOLOGICAL SECTION, SOUTHERN MEDICAL ASSOCIATION
Secretary, Dr. Roy G. Giles, Temple, Texas.

BROOKLYN ROENTGEN RAY SOCIETY
Secretary, Dr. L. J. Taormina, 1093 Gates Ave., Brooklyn, N. Y. Meets monthly on fourth Tuesday, October to April.

BUFFALO RADIOLOGICAL SOCIETY
Secretary-Treasurer, Dr. Joseph S. Gian-Francheschi, 610 Niagara St., Buffalo, N. Y. Meets second Monday of each month except during summer months, place of meeting selected by the host.

CHICAGO ROENTGEN SOCIETY
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FLORIDA STATE RADIOLOGICAL SOCIETY
Secretary, Dr. J. N. Moore, 210 Professional Bldg., Ocala, Florida. Meetings in May and November.

GEORGIA RADIOLOGICAL SOCIETY
Secretary, Dr. R. C. Pendergrass, Prather Clinic Bldg., Americus, Ga. Meets in November and at annual meeting of Medical Association of Georgia in the spring.

ILLINOIS RADIOLOGICAL SOCIETY
Secretary, Dr. Wm. DeHollander, St. John's Hospital, Springfield, Ill. Meetings held quarterly, on the fourth Sunday of the month.

INDIANA ROENTGEN SOCIETY
Secretary, Dr. C. C. Taylor, 23 E. Ohio St., Indianapolis, Ind. Meeting held the second Sunday in May annually.

KENTUCKY RADIOLOGICAL SOCIETY
Secretary, Dr. J. C. Bell, 402 Heyburn Bldg., Louisville. Meets annually in Louisville on third Sunday afternoon in April.

LONG ISLAND RADIOLOGICAL SOCIETY
Secretary, Dr. Marcus Wiener, 1430-48th St., Brooklyn, N. Y. Meets Kings County Med. Soc. Bldg. monthly on fourth Thursday, October to May, 8:30 P.M.

MICHIGAN ASSOCIATION OF ROENTGENOLOGISTS
Secretary, Dr. J. E. Lofstrom, 1536 David Whitney Bldg., Detroit. Three meetings a year, Fall, Winter, Spring.

MILWAUKEE ROENTGEN RAY SOCIETY
Secretary, Dr. I. I. Cowan, Mt. Sinai Hospital, Milwaukee, Wis. Meets monthly on first Friday at University Club.

MINNESOTA RADIOLOGICAL SOCIETY
Secretary, Dr. J. P. Medelman, 572 Lowry Medical Arts Bldg., St. Paul.

NEBRASKA RADIOLOGICAL SOCIETY
Secretary, Dr. D. A. Dowell, Medical Arts Bldg., Omaha, Nebr. Meets third Wednesday of each month, at 6 P.M. at either Omaha or Lincoln.

NEW ENGLAND ROENTGEN RAY SOCIETY
Secretary, Dr. A. O. Hampton, Massachusetts General Hospital, Boston, Mass. Meets monthly on third Friday, Boston Medical Library.

RADIOLOGICAL SOCIETY OF NEW JERSEY
Secretary, Dr. W. J. Marquis, 198 Clinton Ave., Newark. Meets annually at time and place of State Medical Society. Mid-year meetings at place chosen by president.

NEW YORK ROENTGEN SOCIETY
Secretary, Dr. E. J. Ryan, St. Luke's Hospital, New York City. Meets monthly on third Monday, New York Academy of Medicine, at 8:00 P.M.

NORTH CAROLINA ROENTGEN RAY SOCIETY
Secretary, Dr. Major Fleming, Rocky Mount, N. C. Annual meeting at time and place of State Medical Society. Mid-year scientific meeting at place designated.

CENTRAL NEW YORK ROENTGEN RAY SOCIETY
Secretary, Dr. C. F. Potter, 820 S. Crouse Ave., Syracuse. Three meetings a year. January, May, November.

OHIO RADIOLOGICAL SOCIETY
Secretary, Dr. J. E. McCarthy, 707 Race St., Cincinnati. Meets at time and place of annual meeting of Ohio State Medical Association.

PACIFIC ROENTGEN SOCIETY
Secretary, Dr. L. H. Garland, 450 Sutter St., San Francisco, Calif. Meets annually, during meeting of California Medical Association.

PENNSYLVANIA RADIOLOGICAL SOCIETY
Secretary, Dr. L. E. Wurster, 416 Pine St., Williamsport Pa. Next annual meeting, William Penn Hotel, Pittsburgh, Pa., May 16–17, 1941.

PHILADELPHIA ROENTGEN RAY SOCIETY
Secretary, Dr. B. R. Young, Temple University Hospital. Meeting first Thursday of each month from October to May inclusive, at 8:15 P.M., in Thompson Hall, College of Physicians, 19 S. 22d St.

PITTSBURGH ROENTGEN SOCIETY
Secretary, Dr. H. W. Jacox, 4800 Friendship Ave. Meets second Wednesday each month, 4:30 P.M., October to June, Pittsburgh Academy of Medicine.

ROCHESTER ROENTGEN RAY SOCIETY, ROCHESTER, N. Y.
Secretary, Dr. S. C. Davidson, 277 Alexander St. Meets on second Thursday from October to May, inclusive, 8 P.M., Rochester Academy of Medicine Building.

* Secretaries of Societies not here listed are requested to send the necessary information to the Editor.

St. Louis Society of Radiologists
Secretary, Dr. W. K. Mueller, University Club Bldg. Meets fourth Wednesday of October, January, March and May, at a place designated by the president.

San Francisco Radiological Society
Secretary, Dr. H. A. Hill, 450 Sutter St., San Francisco. Meets monthly on third Thursday at 7:45 P.M., first six months at Toland Hall, second at Lane Hall.

South Carolina X-Ray Society
Secretary, Dr. T. A. Pitts, Baptist Hospital, Columbia, S. C. Meets in Charleston on first Thursday in November, also at the time and place of South Carolina State Medical Association.

Tennessee Radiological Society
Secretary, Dr. F. B. Bogart, 311 Medical Arts Bldg., Chattanooga, Tenn. Meets annually at the time and place of the Tennessee State Medical Association.

Texas Radiological Society
Secretary, Dr. L. W. Baird, Scott and White Hospital, Temple, Texas.

University of Michigan Department of Roentgenology Staff Meeting
Meets each Monday evening from September to June, at 7 P.M. at University Hospital.

University of Wisconsin Radiological Conference
Secretary, Dr. E. A. Pohle, 1300 University Ave., Madison, Wis. Meets every Thursday from 4:00–5:00 P.M., Room 301, Service Memorial Institute.

Virginia Radiological Society
Secretary, Dr. C. H. Peterson, 603 Medical Arts Bldg., Roanoke, Va. Meets annually in October.

Washington State Radiological Society
Secretary, Dr. K. J. Holtz, American Bank Bldg., Seattle. Meets fourth Monday of each month at the College Club, Seattle.

X-Ray Study Club of San Francisco
Secretary, Dr. J. M. Robinson, University of California Hospital. Meets monthly in evening on third Thursday.

Cuba

Sociedad de Radiología y Fisioterapia de Cuba
President, Dr. J. Manuel Viamonte, Hospital Mercedes, Habana, Cuba. Meets monthly in Habana.

British Empire

British Institute of Radiology Incorporated with the Röntgen Society
Meets monthly on third Thursday from November to June inclusive, at 8:15 P.M., 32 Welbeck St., London.

Section of Radiology of the Royal Society of Medicine (Confined to Medical Members)
Meets on the third Friday of each month during the winter at 8:15 P.M. at the Royal Society of Medicine, 1, Wimpole St., London, W. 1.

Faculty of Radiologists
Secretary, Dr. M. H. Jupe, 32 Welbeck St., London, W. 1, England.

Section of Radiology and Medical Electricity, Australasian Medical Congress
Secretary, Dr. H. M. Cutler, 139 Macquarie St., Sydney, New South Wales.

Radiological Section of the Victorian Branch of the British Medical Association
Secretary, Dr. Keith Hallam, St. George's Hospital, K.E.W., Melbourne, E. 4, Victoria, Australia. Meets monthly from March to November inclusive for scientific discussion.

Canadian Association of Radiologists
Secretary, Dr. A. C. Singleton, Medical Arts Bldg. Toronto, 5, Ontario.

Section of Radiology, Canadian Medical Association
Secretary, Dr. C. M. Jones, Inglis St., Ext., Halifax, N.S.

Radiological Section, New Zealand British Medical Association
Secretary, Dr. Colin Anderson, Invercargill, New Zealand. Meets annually.

Continental Europe

Belgian Society of Roentgenology
Secretary, Dr. J. Boine, Avenue des Alliés, 134, Louvain (Belgium). Meets monthly on second Sunday at d'Egmonds Palace, Brussels, except in the summertime.

Sociedad Espanola de Radiologia y Electrologia
Secretary, Dr. J. Martin-Crespo, Fuencarral, 7. Madrid, Spain. Meets monthly in Madrid.

Société de Radiologie Médicale de France
Meets monthly on second Tuesday, except during months of August and September, 12 Rue de Saine, Paris.

Société Suisse de Radiologie (Schweizerische Röntgen-Gesellschaft)
Secretary for French language, Dr. A. Grosjean La Chaux de Fonds.
Secretary for German language, Dr. Scheurer, Molzgasse, Biel. Meets annually in different cities.

Société Francaise d'Electrothérapie et de Radiologie Médicale
Meets monthly on fourth Tuesday, except during months of August and September, 12 Rue de Saine, Paris.

Association of German Roentgenologists and Radiologists in Czecho-Slovakia
Secretary, Dr. Walter Altschul, German University, Prague, 11.52.

Deutsche Röntgen-Gesellschaft (Gesellschaft für Röntgenkunde und Strahlenforschung)
Meets annually in April, alternating one year in Berlin, one year in some other German city. Meets in addition every two years with the Gesellschaft deutscher Naturforscher und Aertze.
Permanent Secretary, Professor Dr. Haenisch, Klopstockstrasse 10, Hamburg, Germany.

Süd- und Westdeutsche Röntgengesellschaft
Meets annually in different cities.

Nord- und Ostdeutsche Röntgengesellschaft
Meets annually in different cities.

Dutch Society of Electrology and Roentgenology
Holds two meetings a year in Amsterdam, one in the spring, and one in the fall.

Societa Italiana Radiologia Medica
Secretary, M. Ponzio, University of Turin, Prof. Turin.

Societatea Romana de Radiologie si Electrologie
Secretary, Dr. Oscar Meller, Str. Banul Mărăcine, 30, S. I., Bucuresti, Roumania. Meets second Monday in every month with the exception of July and August.

All-Russian Roentgen Ray Association, Leningrad, USSR in the State Institute of Roentgenology and Radiology, 6 Roentgen St.
Secretaries, Drs. S. A. Reinberg and S. G. Simonson. Meets annually.

Leningrad Roentgen Ray Society
Secretaries, Drs. S. G. Simonson and G. A. Gusterin. Meets monthly, first Monday at 8 o'clock State Institute of Roentgenology and Radiology, Leningrad.

Moscow Roentgen Ray Society
Secretaries, Drs. L. L. Holst, A. W. Ssamygin and S. T. Konobejevsky. Meets monthly on first Monday at 8 o'clock.

Scandinavian Roentgen Socieities
The Scandinavian roentgen societies have formed a joint association called the Northern Association for Medical Radiology, meeting every second year in the different countries belonging to the Association. Each of the following societies, with exception of the Denmark Society, meets every second month except in the summertime.

Society of Medical Radiology in Sweden
Meets in Stockholm.

Society of Medical Radiology in Norway
Meets in Oslo.

Society of Medical Radiology in Denmark
Secretary, Dr. G. Biering, Copenhagen. Meets the second Wednesday of each month from October to July in Copenhagen, at 8 o'clock in the State Institute of Roentgenology.

Society of Medical Radiology in Finland
Meets in Helsingfors.

Vienna Roentgen Society
Meets first Wednesday of each month, at 6:30 P.M. at Zentral-Röntgen Institut des allgemeinen Krankenhauses Alserstrasse 4.

Orient

Japan X-Ray Association
c/o Orthopedic Surgery, Tokyo Imperial University. Meets annually in April.

Kinki Roentgen-Abend Society
Director, Dr. Prof. Taiga Saito, Ogawaoike Tyoto, Japan. Meets bi-monthly on third Sunday.

BULLETIN OF THE INTER-SOCIETY COMMITTEE FOR RADIOLOGY

MASS CHEST SURVEYS*

Like ancient Gaul, the subject of group surveys for the recognition of tuberculosis divides itself naturally into three parts. These are (1) Should such surveys be undertaken? (i.e., are they of sufficient value to the community to warrant the necessary expenditure of time, effort and money?) (2) How, in general, are such surveys made? (i.e., under what auspices, by whom and in what technical fashion?) and (3) How do we think they should be made?

To obtain information that would assist in formulating an answer to each of these questions, the Inter-Society Committee for Radiology has recently surveyed the situation and the preponderance of opinion among qualified observers throughout the country by means of a questionnaire. Replies were received from State representatives of the Committee and Secretaries of regional radiological societies in forty States.

We could easily devote a great deal of time to the discussion of the question, "Should such surveys be undertaken?" Most—or at least many—of us would be inclined to answer in the negative and there is much to support such an answer. When it is remembered that only from 0.25 to 0.50 per cent of children are found to have open cases of tuberculosis and that no means are available for requiring either isolation or treatment of those discovered; when it is remembered that surveys of school children (certainly those most commonly surveyed) ignore the teacher, the cafeteria attendant, and the food handler, one might be justified in considering the method a little extravagant.

A recent issue of the *Journal of the American Medical Association* pointed out that in the eastern part of the country about 60 per cent of patients in tuberculosis sanatoria voluntarily discharge themselves even though bacilli are being found regularly in their sputa. Garland's pertinent comment on this is that it seems futile to make expensive efforts to catch a few open cases on the left hand side of the room and to allow 60 per cent of those you catch to walk out through the right hand side of the room to mingle with and infect the populace at large.

Surveys do, of course, create a group consciousness about tuberculosis. Their chief value undoubtedly is educational. Unfortunately, however, our discussion of this question is a purely academic one since the matter has been settled for us by the various foundations, health agencies, and tuberculosis associations who conduct these surveys, and we can better employ our time in discussion of the second question, "How, in general, are such surveys made?"

By this question we mean to inquire as to the agencies conducting mass surveys; the means by which they are conducted; the medical and particularly the roentgenological personnel; and the medical and again particularly the roentgenological value of these findings.

Questions which particularly concern us are: (1) Should surveys be on a mass or group basis, or performed by radiologists on an individual basis? And (2) How should the radiologist be compensated?

Surveys are conducted by State and local tuberculosis associations; by State and local boards of health, boards of education and similar official and quasi-official bodies. In some instances, industrial insurance carriers or large-scale employers have also undertaken group surveys.

In the majority of instances—at least in those reported to the Committee from various sections of the country—they have been actually made by these agencies just mentioned through paid professional employees and with their own technical equipment.

Some surveys have been made by contract with commercial agencies, notably the Powers Company, and in a few in-

* A report by a special committee to the Board of Chancellors of the American College of Radiology, on February 17, 1941, presented by Dr. L. S. Goin, Chairman.

stances they have been carried out with the cooperation of local radiologists.

The majority of surveys seem to have been made by making ordinary postero-anterior roentgenograms; some have used paper films and a smaller but increasing number employed fluorophotographic methods.

The value of any one of these methods depends absolutely upon two factors: (1) the technical quality of the roentgenograms produced, and (2) the training and experience of the observer who interprets it.

There seems to be no expressed general dissatisfaction with these two factors and apparently and in general the technical quality of roentgenograms produced is at least average, while the training and experience of the observer seems to be considered adequate.

These conclusions are based on statements by radiologists of which the following are random samples:

"The unit includes a physician who is fairly well trained in interpretation of chest films" . . . " . . . an exceptionally well trained man and I think the work was done very well indeed when the circumstances are considered."

"It might be possible to arrange for a radiologist to supervise the interpretations but I doubt . . . if the interpretation would be any more accurate for Dr.———— has had unusually wide experience."

At this point it will be well to recapitulate the conclusions thus far justified by the factual reviews attempted by the Committee, and these conclusions are:

In spite of a fairly widespread feeling that group or mass surveys are more spectacular than useful, they are being made and will continue to be made. Surveys are in most part conducted by official bodies employing their own personnel. Ordinary roentgen films are chiefly used, but fluorophotographic methods are being increasingly employed. It now remains for us to consider the third question: "How do we think surveys should be made?"

An important and frequently overlooked consideration is this: A survey should be—and thoroughly understood to be—a mass survey, and not a series of complete roentgen diagnoses. That is to say that the function of a survey is, or should be, to eliminate the healthy from consideration so that those who need regular roentgen examination of the chest may be identified. If this criterion were constantly in the minds of the survey-makers, we would need have little concern with the details and methods of surveying, but unfortunately "survey" and "regular diagnosis" seem to be pretty well confused and perhaps in straightening out these confused ideas the American College of Radiology can be of real service to the community.

In Bulletin III, the Air Hygiene Foundation devotes an entire page to the prominent display of a statement credited to Pendergrass and Hodes in which they emphasize the necessity of roentgen study of the lungs of those not dismissed as normal by survey methods, and this needs continual emphasis. We may then repeat (commencing to answer our third question) that we think surveys should remain preliminary diagnostic surveys and not regular diagnostic measures.

Undoubtedly the best way to conduct mass surveys of school children or industrial groups would be by cooperation between local radiologists and anti-tuberculosis associations. If the groups to be surveyed could be divided into small subgroups and sent to radiologists' offices, we would have the ideal solution. Failing this ideal, roentgenograms of those included in a survey should be studied by a radiologist or (perhaps better) by a group of radiologists or of radiologists and tuberculosis specialists. Certainly this important work should not be entrusted to the young and unskilled physicians who are ordinarily hired for survey work.

There is, of course, no reason why a radiologist or any other physician should contribute his services to a community venture. This does not mean that the physician is unwilling to cooperate or to carry his share of any burden; it only means that

in any joint project of a community the physician's role is that of a good citizen of like social and financial standing, but that it is as absurd for the community to expect him to donate his services as to expect the same of the film manufacturer, the nurse, the driver of the camion or any other person properly earning his bread by his efforts.

In spite of these obvious facts, however, if the other physicians engaged in such a communal enterprise are giving their services, the radiologist risks setting himself apart from his profession by his refusal to do likewise. Several approaches to the solution of this problem have been attempted.

In Florida the State Board of Education and the State Board of Health advise chest roentgen-ray examinations for all school employees and provide that it may be made by any Doctor of Medicine. The fee is left to private treaty.

In New Jersey the Examiner "may be the school medical inspector or one or more physicians engaged for that purpose," and the State Board of Health rules provide that "x-ray work may be purchased from tuberculosis sanatoria, local general hospitals, local roentgenologists or commercial agencies" and the "purchaser" is urged to inquire of the commercial agencies whether the price quoted includes the reading of the roentgen film.

When such poor comprehension of the problem is encountered, one wonders whether the survey conducted directly by a State Board of Health would not be preferable.

In Monmouth County, New Jersey, however, the tuberculosis committee of the County Medical Society, comprising radiologists and tuberculosis specialists, furnishes interpretations of films made by a survey agency, and this plan is commended.

Your Committee recommends that:

1. The existence of mass surveys as going concerns be recognized as an accomplished fact;

2. That radiologists be urged to lend themselves to such surveys as are truly community efforts on the same terms as their fellow practitioners;

3. That the plan of the Monmouth, New Jersey, County Medical Society be commended;

4. That the American College of Radiology constantly insist on the recognition of the survey as an initial crude diagnostic survey and not as a series of diagnoses.

DERMOID CYSTS OF THE OVARY

To the Editor:

I read your interesting editorial on "Dermoid cysts of the ovary" in the December, 1940, issue of the JOURNAL. I am taking the liberty of sending a reprint of a paper published in the *American Journal of Obstetrics and Gynecology* in August, 1939.[1] The sign afterwards described by Robins and White was demonstrated and correctly interpreted in my publication.

After I completed my paper, I found that the sign of the "radiolucent tumor" had already been described by two Swedish authors, Laurell[2,3] and Odqvist,[4] in *Acta radiologica* in 1922, 1925 and 1926. I quoted their publications in my article and changed the title of my article to "A neglected sign for roentgenologic diagnosis of intra-abdominal dermoid cyst."

To the best of my knowledge, this observation was made independently by Laurell and by Odqvist, by Robins and White, and by myself. However, Laurell was seventeen years and Odqvist thirteen years ahead of us.

GERHARD DANELIUS
Mt. Sinai Hospital
Chicago, Illinois

[1] Danelius, G. A neglected sign for roentgenologic diagnosis of intra-abdominal dermoid cyst. *Am. J. Obst. & Gynec.*, 1939, *38*, 348–350.

[2] Laurell, Hugo. Ein Beitrag zur Röntgenologie der echten Mischgeschwülste des Rumpfes. *Acta radiol.*, 1922, *1*, 406–417.

[3] Laurell, Hugo. Weitere Beiträge zur Röntgenologie der abdominellen Mischgeschwülste. *Acta radiol.*, 1925, *4*, 480–498.

[4] Odqvist, Henning. Beitrag zur Röntgendiagnostik der Bauchtumoren. *Acta radiol.*, 1926, *7*, 253–258.

CONFERENCE OF EASTERN RADIOLOGISTS

The Thirty-first Annual Midwinter Conference of Eastern Radiologists met at the Southern Hotel in Baltimore, Maryland, on January 31 and February 1, 1941. Arrangements for the meeting had been made by the Local Committee consisting of Dr. John W. Pierson, Dr. Marcus Ostro, Dr. Harold E. Wright, Dr. Whitmer B. Firor, Dr. Walter L. Kilby, Secretary and Dr. Henry J. Walton, Chairman.

The Friday morning session was held at the Hurd Memorial Hall, Johns Hopkins Hospital, with Dr. John W. Pierson as Chairman. The following scientific program was presented:

The Significance of X-Ray Findings in Childhood tuberculosis. Miriam Brailey, M.D.

Some Comparative Cytological Effects of X-Rays, Gamma Rays and Neutrons on Living Tumor Cells Maintained in Continuous Culture. George Gey, M.D.

Hypothyroid Epiphyseal Dysgenesis. Lawson Wilkins, M.D.

Rarefying Metabolic Diseases of Bone. John E. Howard, M.D.

The Disease Background of Schüller-Christian's Syndrome. William B. Vandergriff, M.D.

Skeletal Manifestations of the Lymphoblastomata. George J. Farber, M.D.

On Friday afternoon the scientific session was held at Assembly Hall, Broadway and Monument St. Dr. Marcus Ostro was the Chairman and the following papers were presented:

So-called Miliary Pulmonary Shadows. Charles R. Austrian, M.D.

The Dyschondroplasias. Alexander J. Schaffer, M.D.

Syphilitic Bone Lesions. Edwin D. Weinberg, M.D.

Interesting Renal Lesions Diagnosed by Retrograde Pyelography. Benjamin S. Abeshouse, M.D.

Diagnostic X-Ray Methods in Obstetrics Exclusive of Pelvimetry and Cephalometry. Webster H. Brown, M.D.

On Friday evening a banquet was held at 8:30 at the Southern Hotel.

The concluding session was held on Saturday morning at Gordon Wilson Hall, University Hospital, Dr. Henry J. Walton, Chairman. The following scientific program was presented:

Sources of Error in the Roentgen Diagnosis of Colon Lesions. Walter L. Kilby, M.D.

Epidural Spinal Abscess. Maurice C. Pincoffs, M.D.

Chemotherapy and Primary Pneumonia in Infants. Gibson J. Wells, M.D.

Physiological and Pathological Changes Observed in Roentgen Studies of the Urinary Tract. J. Mason Hundley, M.D.

Abnormal Ossifications; Report of Cases. Arthur M. Shipley, M.D.

Treatment of Adamantinomata. Grant E. Ward, M.D., and A. M. Duff, M.D.

Demonstration of Prosthetic Appliances for Intraoral and Superficial Radium Treatments. James E. Pyott, M.D., and G. W. Gaver, M.D.

CONFERENCE OF MID-WESTERN RADIOLOGISTS

The Fifth Annual Clinical Conference of Mid-Western Radiologists was held on February 14 and 15, 1941, at Rochester, Minnesota. The program was given by members of the Staff of the Mayo Clinic and all sessions were held in Plummer Hall, Mayo Clinic Building.

On Friday morning the following scientific program was presented, the Chairmen being Dr. C. Allen Good and Dr. Arthur U. Desjardins:

Roentgen Treatment of Lymphoblastoma in Relation to Certain Important Clinical Features. A. U. Desjardins, M.D.

The Selection of Patients for, and the Surgical Treatment of Cancer of the Uterine Fundus. V. S. Counseller, M.D.

The Results of Radium Treatment of Cancer of the Uterine Fundus with Reference to the Grade of Malignancy of the Lesion. Robert E. Fricke, M.D.

Factors Influencing the Prognosis in Cancer of the Uterine Cervix. Harry H. Bowing, M.D.

The Roentgen Treatment of Functional Menstrual Irregularities in Young Women. Della G. Drips, M.D., and E. T. Leddy, M.D.

Roentgen Pelvimetry: Its Value in Selected Obstetrical Cases. A. B. Hunt, M.D., and C. Allen Good, M.D.
Some Physical Factors Affecting the Quality and Quantity of Radiation. M. M. D. Williams, Ph.D.
Urographic Findings in Essential Hypertension. W. F. Braasch, M.D.
Significance of Urologic Lesions in the Treatment of Hypertension. N. W. Barker, M.D.
Small Malignant Lesions of the Stomach
Clinical Features. A. M. Snell, M.D.
Roentgenologic Features. B. R. Kirklin, M.D.
Surgical Treatment. Howard K. Gray, M.D.
Pathology. Wm. C. MacCarty, Sr., M.D.

The Chairmen of the Friday afternoon session were Dr. Robert E. Fricke and Dr. Harry M. Weber. The scientific program was continued with the following papers:

Tumors of the Urinary Bladder
Urologic Features. E. N. Cook, M.D.
Surgical Treatment. J. T. Priestley, M.D.
Roentgen Treatment. W. C. Popp, M.D.
Prostatic Calculi. G. J. Thompson, M.D.
Changing Roentgenologic Concepts of Renal Tuberculosis. J. L. Emmett, M.D.
The Effect of Altitude on Certain Surgical and Medical Conditions. W. R. Lovelace, M.D., H. C. Hinshaw, M.D., and C. Allen Good, M.D.
Actinomycosis of the Face and Neck. Gordon B. New, M.D., and A. U. Desjardins, M.D.
Acute Sinusitis: Its Treatment with Roentgen Rays. H. L. Williams, M.D., and W. C. Popp, M.D.
The Diagnosis and Treatment of Leucosarcoma. C. H. Watkins, M.D., and W. C. Popp, M.D.
The Selection and Arrangement of Material for Lantern Slides. A. B. Nevling, M.D., and R. M. Hewitt, M.D.
A Two-way Tilt Fluoroscopic Table. J. D. Camp, M.D.
Presentation of Interesting or Unusual Cases. Members of Mayo Clinic Staff.

On Friday evening a banquet was held in the Hotel Kahler.

The scientific program was resumed on Saturday morning, Dr. John D. Camp and Dr. E. T. Leddy, Chairmen. The following papers were presented:

Dental Roentgenography in Systemic Diseases. Louis T. Austin, D.D.S., and E. C. Stafne, D.D.S.
Platybasia. M. N. Walsh, M.D., W. McK. Craig, M.D., and John D. Camp, M.D.
Epidermoid Tumors of the Skull. J. Grafton Love, M.D., J. W. Kernohan, M.D., and John D. Camp, M.D.
Calcified Basal Nuclei. L. M. Eaton, M.D., and John D. Camp, M.D.
Studies of Intestinal Activity. F. C. Mann, M.D.
Experiences with Intestinal Intubation. E. G. Wakefield, M.D., and M. T. Friedell, M.D.
Forms of Ulcerative Colitis
From the Standpoint of the Clinician. J. A. Bargen, M.D.
From the Standpoint of the Proctologist. N. D. Smith, M.D.
From the Standpoint of the Radiologist. H. M. Weber, M.D.
Polypoid Lesions of the Colon in Children
Clinical Features. R. L. J. Kennedy, M.D.
Surgical Treatment. C. F. Dixon, M.D.
Technic of Roentgenologic Examination. H. M. Weber, M.D.

The scientific program was concluded on Saturday afternoon, Chairmen Dr. Harry H. Bowing and Dr. Walter C. Popp, and the following papers were presented:

Multiple Osteochondritis. H. H. Young, M.D.
Brucellosis: Skeletal Manifestations. H. B. Macey, M.D.
Hemangioma of the Vertebrae. R. K. Ghormley, M.D., A. W. Adson, M.D., and A. U. Desjardins, M.D.
Tumors of Bone
Surgical Pathology. A. C. Broders, M.D.
Roentgen Treatment. A. U. Desjardins, M.D.
Roentgenologic Features. C. G. Sutherland, M.D.
Experimental Studies on the Production of the Silicotic Lesion. W. S. Lemon, M.D.
Adhesive Pericarditis. A. R. Barnes, M.D., and S. W. Harrington, M.D.
Intrapulmonary Lesions
Clinical and Bronchoscopic Features. H. J. Moersch, M.D.
The Importance of Roentgen Examination. B. R. Kirklin, M.D.
Roentgen Treatment. E. T. Leddy, M.D.
Surgical Treatment. S. W. Harrington, M.D.

On Saturday evening guests were invited to visit the Mayo Foundation Museum of

Hygiene and Medicine and members of the Radiologic Staff were present to demonstrate the radiologic exhibits in the Museum. At the Mayo Foundation House Dr. C. G. Sutherland gave a "Demonstration of Roentgenograms Reduced on Microfilm for Teaching Purposes."

X-RAY STUDY CLUB

The X-ray Study Club of San Francisco meets in the evening of the third Thursday of every month for the discussion of cases of radiological interest, the diagnoses of which have been proved. This organization was originally founded by the Staffs of the Radiology Department of Stanford University and of the University of California. In recent years, it has been enlarged to include the radiologists of the San Francisco Bay area. All radiologists who may be in San Francisco at the time are cordially invited to attend. They should communicate with the secretary, Dr. J. M. Robinson at the University of California Hospital, concerning the place of meeting which is changed every four months.

PENNSYLVANIA RADIOLOGICAL SOCIETY

The annual meeting of the Pennsylvania Radiological Society, which will be its twenty-fifth anniversary, will be held at the William Penn Hotel, Pittsburgh, Pennsylvania, May 16 and 17, 1941. The guest speakers will be Dr. Ross Golden of New York, whose subject will be "Lesions of the Gastrointestinal Tract" and Dr. John T. Murphy of Toledo, Ohio, whose subject will be "The Treatment of Superficial Malignancies." Roentgenologists from other societies are cordially invited to attend.

BOOK REVIEWS

Books sent for review are acknowledged under: Books Received. This must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers as space permits.

Hugh Young: A Surgeon's Autobiography. Cloth. Price, $5.00. Pp. 554, with over 100 drawings by William P. Didusch and 3 color prints. New York: Harcourt, Brace and Company, 1940.

It must have been a memorable day in the life of a young physician when he briskly turned off the long corridor of the Johns Hopkins Hospital on a beautiful autumn day in 1897, and almost collided with the austere Professor of Surgery, William Stewart Halsted. The budding medico was Hugh Hampton Young, just turned twenty-seven years, a recent graduate of the Medical School of the University of Virginia who had been associated with the Johns Hopkins Hospital for a relatively brief period and apparently in a purely tentative capacity. Halsted said: "Don't apologize, Young. I was looking for you, to tell you we want you to take charge of the department of genito-urinary surgery." The reply was: "This is a great surprise. I know nothing about genito-urinary surgery." Whereupon Dr. Halsted answered, "Welch and I said you didn't know anything about it, but we believe you can learn." All of this sounds like a very casual occurrence and an unimportant one, as related in Hugh Young's autobiography. It was neither. In the first place, it marks the beginning of the career of the most eminent urologist of the United States, and one who has an outstanding international reputation. An estimate of his place in the profession is evidenced by the recent statement of Dr. William C. Quimby, which was made on presenting the Keyes medal to Dr. Young, as follows: "We believe that the services you have rendered to mankind through your noble work have not been surpassed by any other American in the whole field of medicine." Second, the incident is of interest because it was so typical of Halsted to appear to be so unconcerned about a momentous decision, whereas actually every move of his was preceded by careful deliberation and forethought. And, finally, the "Professor" was so frank as to say "Welch and I." As the history of the Johns Hopkins Hospital and Medical School unfolds to succeeding generations, this combination will be recognized as one of the greatest influences in the development of that institution. The Welch part remains constant while the "I" includes many of the members of the staff of the Hospital and faculty of the Medical School. This and many similar incidents of great human interest go to make up one of the reasons why Hugh Young's autobiography is an important historic document and one which holds the interest of the reader from the first to the last page. The author has had a useful, gratifying and fascinating life, and, moreover, he can tell a good story. These are the desiderata of a good autobiography.

When a professional man writes an autobiography, and there have been a number by physicians recently, one wonders whether it will be appreciated only by his friends, or by those who have a slight acquaintance with him but a great interest in his environment and associations, or, finally, whether his literary effort will appeal to a wide variety of readers. When Hugh Young's book is considered from these standpoints, it is the reviewer's opinion that it will be regarded as entertaining and informative by a great majority of men, women and adolescents who read it.

The story of the development of genito-urinary surgery is well told. When Hugh Young took over the primitive beginnings of the department in Baltimore in 1897, there were only two crude cystoscopes available of the original type which had been invented by Max Nitze in 1876. Young has observed the development of all the modern diagnostic aids in the study of abnormalities of the bladder, ureters and kidneys, which have given such enviable exactness to the recognition of pathologic changes in that part of the body. The "phthalein test" which was the first practical method of determining renal function, matured under his eyes after Ira Remsen, then Professor of Chemistry at Johns Hopkins University, discovered it in 1884, and John J. Abel, Professor of Pharmacology observed that it was eliminated by the kidneys. A first-hand account in simple language is given of the development of surgery of the prostate gland. The various steps are

illustrated with clear and accurate drawings which for the most part are made by William P. Didusch, the outstanding medical artist, who has long been associated with Dr. Young. Bladder tumors are also discussed, as are urinary calculi, genito-urinary tuberculosis, and his most careful studies on hermaphroditism about which he has made extensive studies and has become the leading authority.

His experiences with the development of the mercurial antiseptic drug, mercurochrome, are recounted and the history of its synthesis is told. His interest in this subject, in which he has persisted since the beginning, arose as a result of hearing Paul Ehrlich discuss salvarsan and other forms of chemotherapy at the International Medical Congress in London in 1913. Hugh Young's continued enthusiasm for experimental studies and clinical observations on the effects of such chemicals has been a stimulus to all workers in this field, for which he should be given well deserved recognition.

His experiences in the World War of 1917 were unusual and are of special interest at the present time. He crossed the Atlantic with General Pershing and his Staff on the Steamer *Baltic* which left New York on May 29, 1917, and was, therefore, among the earliest United States Army personnel in Europe. His purpose in going was to operate on special urologic cases in the British hospitals and to study the progress which had been made in urology and venereology in the armies of the Allies. After a brief stay in England, where he studied the venereal problem, he was sent to France and there, as Director of the Division of Urology of the United States Army, was put in charge of all urologic and venereal disease work. The venereal disease program of the American Expeditionary Forces, as directed by Dr. Young, is related in detail and makes especially appropriate reading at a time such as this. All of his war experiences, both professional and personal, are entertainingly presented.

Hugh Young's versatility is illustrated by his interest in bringing to light the justifiable prior claim of Crawford W. Long, a Physician of Danielsville, Georgia, as having been the first to use ether as an anesthetic for a surgical operation. This was four years before the historic operation under ether at the Massachusetts General Hospital in Boston when John Collins Warren made the classic remark, "Gentlemen, this is no humbug." The reviewer recalls having read Young's article in the *Johns Hopkins Hospital Bulletin* when he was a third year medical student, in which Dr. Long's claims are stated. He could not refrain from pondering over the reason why the busy Professor of Genito-Urinary Surgery, who was known to him only from afar, should interest himself on behalf of an obscure practitioner of Georgia, in a historic medical controversy which seemed to be so remote from such practical things as removing prostate glands and related operations. For the lack of a better explanation he finally concluded naïvely, without troubling to ascertain the facts, that it must have been "because they were both Southerners." In a sense this was true, because Dr. Young states that while on a visit to his home in Texas he met Mrs. Fannie Long Taylor, the daughter of Dr. Long, who informed him of her father's claim to the discovery of ether as an anesthetic. The proof of Dr. Long's assertions were eagerly examined and on returning to Baltimore, after being convinced that the claims were valid, he consulted with Dr. Halsted who advised that the data be presented before the Johns Hopkins Medical Society. This was done in the fall of 1896 and the material later appeared in the *Johns Hopkins Hospital Bulletin*. Although undoubtedly Long was the first to use ether as a surgical anesthetic, as Welch says, "we cannot assign to him any influence upon the historical development of our knowledge of surgical anaesthesia or any share in its introduction to the world at large." Nevertheless it must be admitted that Hugh Young gave publicity to the righteous claims of the one who originally administered the drug, and one who had hitherto been ignored.

Throughout his life Hugh Young has had an unusual opportunity to meet and associate with prominent men, both in the profession and in public life. Perhaps one of the most remarkable of these in many respects was James Buchanan Brady ("Diamond Jim"). Of him Young says, "He was indeed one of the most extraordinary men I have ever known,—a rough diamond, it might be said, brought up in the Bowery, self-educated, with unusually flashy tastes, but one of the most persuasive men in America . . . to me it is a great satisfaction to have known him so well." Brady first presented himself to Dr. Young in 1912 with a collection of diseases which, when listed, sound like the minutiae of anatomical diagnoses following a necropsy.

There was nothing insignificant, however, about the pathological changes in his case, which were listed as follows: "Diabetes, Bright's disease, generalized urinary infection, inflammation and obstruction of the prostate gland, difficulty and frequency of urination, angina pectoris and hypertension." He had been told by others that he could not undergo an operation safely on account of the associated heart disease. Young operated upon him successfully, however, with the prostatic punch instrument, under local anesthesia. As evidence of his gratitude, Brady gave a large sum of money to establish the James Buchanan Brady Urological Institute in connection with the Johns Hopkins Hospital and Medical School. This Institute was dedicated on May 4, 1915, with formal ceremonies at which Dr. Edward L. Keyes, Sr., of New York, delivered an address on "Opportunity." All of this is to be found in the autobiography, but nothing is said about the rather extravagant and laudatory remarks of "Popsy" Welch on this occasion, which obviously pleased and at the same time bewildered the remarkable "Diamond Jim." Upon the conclusion of this speech, the blushing donor was undecided as to whether he should stand up and bow or remain seated,—to speak or remain silent. He finally compromised after what seemed to be an interminable period of indecision by arising, bowing stiffly and rather awkwardly in the direction of the accomplished and practiced panegyrist, and then subsiding promptly in his chair. All of this was observed and remembered clearly by the reviewer who, as a second year medical student at "Hopkins," happened to attend casually the dedicatory ceremonies in the old amphitheater off Monument Street, without having the faintest idea of what it was all about.

Hugh Young is to be congratulated, for he has related the story of his life interestingly, honestly and modestly. In it there has been no more of the personal pronoun "I" than is absolutely necessary, and, when all is considered, he has accomplished a great deal about which any man could easily have a justifiable egotism.

C. C. Sturgis

A Textbook of Neuro-radiology. By Cecil P. G. Wakeley, D.Sc., F.R.C.S., F.R.S.E., F.A.C.S., F.R.A.C.S.(Hon.), Fellow of King's College, London, Senior Surgeon, King's College Hospital and West End Hospital for Nervous Diseases, etc., and Alexander Orley, M.D., D.M.R.E., Fellow of the British Association of Radiologists; Radiologist, West End Hospital for Nervous Diseases, etc. Cloth. Price, $8.00. Pp. 336, with 229 illustrations. Baltimore: William Wood and Company, 1938.

Collaborating the works of a surgeon and radiologist, Wakeley and Orley present a bird's-eye view of the roentgenological findings in all types of neurologic lesions. Beginning with the normal anatomy of the skull and the different special projections required for thorough roentgenological study, chapters dealing with skull deformities, injuries, inflammatory lesions, metabolic diseases and neoplasms follow in logical sequence. Separate chapters are devoted to the findings in increased intracranial pressure and those types of intracranial lesions which present a characteristic appearance on films without contrast material. Cerebral arteriography and venography, ventriculography and encephalography complete the portion of the book devoted to the skull. Lesions of the cord and spine, their identification with opaque substance, and a brief chapter on neuropathic disturbances complete the volume.

American roentgenologists would probably not select as a method of choice some of the procedures recommended in this text. For example, cisternal puncture is advised for myelography in preference to the usual lumbar injection. Ventriculography with iodized oil and thorotrast is given so much space that the impression is gained that these are common procedures.

The line drawings are technically excellent. The reproductions are very good, but there is an admixture of them in positive and negative prints. A good bibliography is appended.

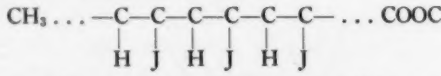
For the general roentgenologist who wishes a survey text on neuro-radiology, this is a useful book.

Morton Helper

Hepatolienographie mit Jodsol. Von Dr. Franz Beckermann, Sekundärarzt der Klinik. Aus der I. medizinischen Klinik der Hansischen Universität (Direktor: Prof. H. H. Berg). Cardboard. Price, RM. 8. Pp. 88, with 65 illustrations. Leipzig: Georg Thieme, 1940.

Since visualization of the spleen and liver in the roentgenogram by means of intravenous injection of thorium dioxide is considered by

many clinicians as fraught with danger, Degkwitz and his co-workers developed a contrast medium which does not contain any radioactive material. The opaque substance is iodine and the solution used is an iodine sol where stearin acid serves as the vehicle:



It contains about 50 per cent solids, 47.5 per cent of which represent the contrast medium and 2.5 per cent protective colloids. Experiments have shown that iodine is almost exclusively excreted in the urine and only a small portion by way of the intestines; the process is usually complete on the seventh day after injection. The amount to be injected depends on the body weight; by multiplying the weight in kilograms by 1.4 the number of cubic centimeters to be administered is obtained. It is important to test every patient first as to his reaction to iodine because some persons are sensitive to it and contraindicate the injection. The technique of administration, the exposure factors, reactions of the patient which sometimes occur and their treatment are outlined. The normal roentgenogram of spleen and liver as well as the roentgenograms of a number of pathological conditions are discussed in detail. That the method has its limitations is clearly stated. At present it seems to be of value in determining the shape, size, and position of spleen and liver, in verifying suspected metastases, in supporting a diagnosis of cholangitis and in definitely establishing cirrhosis of the liver before the presence of clinical symptoms. The illustrations which accompany the text are good and instructive.

Ernst A. Pohle

Electrocardiographic Patterns: Their Diagnostic and Clinical Significance. By Arlie R. Barnes, M.D., Mayo Clinic, Rochester, Minnesota. Cloth. Price, $5.00. Pp. 197, with 95 illustrations. Springfield, Illinois: Charles C Thomas, 1940.

Electrocardiography has made such great strides that the worker in this field must possess a library on this subject. Barnes, one of the keen observers in electrocardiography, has written an excellent review of the experimental and clinical aspects of the subject and even though the records of the chest leads are taken in the old way the book is warmly recommended. The chapters on myocardial infarction, pericarditis, and pulmonary embolism are especially illuminating. The typography and make-up of this book are excellent. The work should be in every electrocardiographic laboratory.

Harold Feil

Reports on Medical Progress, 1939, as Published in the *New England Journal of Medicine*. Compiled and Edited by Robert N. Nye, M.D. Cloth. Price, $5.00, Pp. 562. Boston: Little, Brown and Company, 1940.

This volume is unique in that it attempts to take the place of a year book which covers the entire field of medicine. Peculiar as that may sound, the idea is not too far fetched for the volume contains chapters written by a host of well-known contributors who are outstanding in their fields and who in each case write about their particular field. These contributors in a very concise manner review the present status of their subject and the new developments in each one. These reports were published in the *New England Journal of Medicine* and are now set together in a complete volume. The chapters contain a large number of salient facts and late developments in the diagnosis and treatment of disease. For a specialist who can keep in touch with his own field only during the year, such a book as this is of inestimable value to keep him in touch with what is going on in other fields than his own. The editor and contributors are to be congratulated on a very excellent volume.

Paul C. Swenson

BOOKS RECEIVED

The Head and Neck in Roentgen Diagnosis. By Henry K. Pancoast, M.D., Late Professor of Radiology and Director of the Department of Radiology, University of Pennsylvania; Eugene P. Pendergrass, M.D., Professor of Radiology and Director of the Department of Radiology, University of Pennsylvania; and J. Parsons Schaeffer, M.D., Ph.D., Professor of Anatomy and Director of the Daniel Baugh Institute of Anatomy, Jefferson Medical College. Cloth. Price, $12.50. Pp. 976, with 1251 illustrations. Springfield, Illinois; Charles C Thomas, 1940.

A Handbook of Radiography. By John A. Ross, M.A.(Camb.), M.R.C.S.(Eng.), L.R.C.P.(Lond.), D.M.R.E.(L'pool), Radiol-

ogist, Warrington Infirmary; Visiting Radiologist, Borough General Hospital, Warrington; Clinical Assistant, X-Ray Dept. Royal United Hospital, Liverpool; Visiting Radiologist, Newton Memorial Hospital, Lancs. Cloth. Price, 7s.6d. Pp. 126, with 67 illustrations. London: H. K. Lewis & Co., Ltd., 1940.

The Displacement Method of Sinus Diagnosis and Treatment. By Arthur W. Proetz, A.B., M.D., Professor of Clinical Otolaryngology in the Washington University School of Medicine. Second edition. Cloth. Price, $6.00. Pp. 296, with 157 illustrations. St. Louis: Annals Publishing Co., 1939.

Tomography. By J. B. McDougall, M.D. (Glasg.), F.R.C.P.(Ed.), F.R.S.E., Fellow of the Royal Faculty of Physicians and Surgeons, Glasgow; Adviser in Respiratory Diseases to the British Legion; Medical Director, Preston Hall, Maidstone, Kent; Medical Superintendent, Douglas House, Bournemouth. Cloth. Price, 21/-. Pp. 73, with 110 illustrations. London, H. K. Lewis & Co., Ltd., 1940.

Magenphysiologie für Röntgenzweke: Grundlagen einer physiologischen Deutung des Röntgenbefundes. Von Dr. med. habil. G. A. Weltz, Röntgenfachartz, Dozent und Abteilungsleiter am physiologischen Institut der Universität München. Cardboard. Price, RM. 12. Pp. 77, with 62 illustrations. Leipzig: Georg Thieme, 1940.

Age Morphology of Primary Tubercles. By Henry C. Sweany, M.D., Medical Director of Research, Municipal Tuberculosis Sanitarium, Chicago, and Research Associate, Department of Physiology, University of Chicago. Cloth. Price, $5.00. Pp. 265, with 73 plates. Springfield, Illinois: Charles C Thomas, 1941.

The Choice of Methods of Roentgen Examination of the Chest for Diagnostic Surveys. Medical Series, Bulletin No. III. By Air Hygiene Foundation of America, Inc. Paper. Pp. 73, with 19 illustrations. Pittsburgh, Pa.: Air Hygiene Foundation of America, Inc., 1940.

The 1940 Year Book of Radiology. *Diagnosis:* Edited by Charles A. Waters, M.D., Associate in Roentgenology, Johns Hopkins University; Assistant Visiting Roentgenologist, Johns Hopkins Hospital; Associate Editor, Whitmer B. Firor, M.D., Assistant in Roentgenology, Johns Hopkins University; Assistant in Roentgenology, Johns Hopkins Hospital. *Therapeutics:* Edited by Ira I. Kaplan, B.Sc., M.D., Director, Radiation Therapy Department, Bellevue Hospital, New York City; Associate Radiologist, Lenox Hill Hospital, New York City; Clinical Professor of Surgery, New York University Medical College. Cloth. Price, $5.00. Pp. 496, with 497 illustrations. Chicago: The Year Book Publishers, Inc., 1940.

Radiologic Physics. By Charles Weyl, S. Reid Warren, Jr., and Dallett B. O'Neill, Moore School X-Ray Laboratory, Moore School of Electrical Engineering, University of Pennsylvania. With a foreword by Eugene P. Pendergrass, M.D., Director of the Department of Radiology, University of Pennsylvania. Cloth. Price, $5.50. Pp. 459, with 158 illustrations. Springfield, Illinois: Charles C Thomas, 1941.

Radiology Physics. An Introductory Course for Medical or Premedical Students and for all Radiologists. By John Kellock Robertson, F.R.S.C., Professor of Physics, Queen's University, Kingston, Canada. Cloth. Price, $3.50. Pp. 270, with 188 illustrations. New York: D. Van Nostrand Company, Inc., 1941.

Modern Physics of Roentgenology: For Physicians, Pre-Medical Students, Students of Physics, X-Ray Engineers and X-Ray Technicians. By H. M. Muncheryan, M.Sc., Professor of Physics, California College of Medical Technicians; Author of "A Laboratory Manual of X-Ray Physics." Foreword by Dr. Albert Soiland, M.D., Director of the Los Angeles Tumor Institute. Second edition, revised and enlarged. Cloth. Pp. 391, with 195 illustrations. Los Angeles: Wetzel Publishing Co., Inc., 1940.

The Injured Back and Its Treatment. Edited by John D. Ellis, M.D. Cloth. Price, $5.50. Pp. 377, with numerous illustrations. Sprinfield, Illinois: Charles C Thomas, 1940.

Pathology. By Eugene C. Piette, M.D., Pathologist and Director of the Clinical Laboratories of the West Suburban Hospital, Oak

Park, Illinois; formerly Consultant Pathologist, Chicago State Hospital and Assistant Professor of Pathology, College of Medicine, University of Illinois, Chicago, Ill. Third edition. Cloth. Price, $2.00. Pp. 247, with 60 illustrations. Philadelphia: F. A. Davis, Co., 1940.

Die Entzündungsbestrahlung. Von Dr. med. habil. R. Glauner, Dozent für Röntgenologie an der Universität Köln, Oberarzt am Röntgen- und Lichtinstitut Bürgerhospital. Cardboard. Price, RM. 15. Pp. 190, with 14 illustrations. Leipzig: Georg Thieme, 1940.

The Rôle of the Liver in Surgery. By Frederick Fitzherbert Boyce, B.S., M.D., Diplomate of the American Board of Surgery; Fellow of the American College of Surgeons; Visiting Surgeon: Charity Hospital of Louisiana at New Orleans, French Hospital, Mercy Hospital, Hotel Dieu, Southern Baptist Hospital and Touro Infirmary. Cloth. Price, $5.00. Pp. 365, with 44 illustrations. Springfield, Illinois: Charles C Thomas, 1941.

Hugh Young: A Surgeon's Autobiography. Cloth. Price, $5.00. Pp. 554, with over 100 drawings by William P. Didusch and 3 color prints. New York: Harcourt, Brace and Company, 1940.

Estudos Cirurgicos. 3.ª serie. By Eurico Branco Ribeiro, Director do Sanatorio São Lucas. Cloth. Pp. 219, with 23 illustrations. São Paulo, Brasil: Sociedade Editora Medica Limitada, 1940.

Pesquiza da alça jejunal em cirurgia gastrica. By Dr. Eurico Branco Ribeiro, Director do Sanatorio São Lucas. Paper. Pp. 59, with 15 illustrations. São Paulo, Brasil: Sociedade Editora Medica Limitada, 1940.

Taber's Cyclopedic Medical Dictionary: Including a Digest of Medical Subjects. By Clarence Wilbur Taber, Author, Taber's Dictionary for Nurses, The Cumulative Atlas of the Human Body, Taber's Dietetic Charts, Dictionary of Food and Nutrition, etc., and Associates. Cloth. Pp. 1500, with 273 illustrations. Philadelphia: F. A. Davis Co., 1940.

Graduate Medical Education. Report of the Commission on Graduate Medical Education. Cloth. Pp. 304. Chicago: University of Chicago Press, 1940.

Vol. 45, No. 4

DEPARTMENT OF TECHNIQUE

Department Editor: Robert B. Taft, M.D., B.S., M.A., 105 Rutledge Ave.
Charleston, S. C.

ROENTGENOGRAMS OF THE MANDIBULAR CONDYLE AND ZYGOMATIC ARCH

By FREDERICK R. McGRAIL,* D.D.S., *and* JOSEPH A. DOHERTY, D.M.D.

BOSTON, MASSACHUSETTS

FRACTURES of the facial bones rank high in frequency among the most commonly observed bone injuries. This tendency to fracture may be attributed to an anatomical prominence which renders them susceptible to various kinds of trauma. The automobile, sports and the clenched hand are the important etiologic factors producing injury. Because of the presence of teeth, accuracy in reduction is most essential in order that malocclusion with its attendant interference with normal mastication will not follow. Functional and cosmetic requirements can be fulfilled only when adequate diagnostic measures are employed. In previous articles[1,2] dealing with the management of fractures of the maxillary and malar bones the necessity for a complete reoentgenographic survey has been emphasized.

GENERAL CONSIDERATIONS

The increase in the number of fractures of the condyle of the mandible is the natural result of trauma to the region of the chin induced principally by passengers being thrown against the windshield of the moving automobile. Injury to the condyle may involve any part of it; most often the fracture line runs through the neck of the process. Because of the strong pull of the external pterygoid muscle, displacement of the condylar head usually occurs. Marked displacement invariably involves a distinct interference with the natural relationship of the upper and lower jaws with deviation of the mandible toward the side of injury. Reduction and immobilization is a mechanical and sometimes surgical problem based upon accurate knowledge as to the location and extent of the injury. Examination of the body and lower part of the ramus of the jaw by means of the roentgen ray presents no unusual difficulties. However, the determination of the degree of involvement of the condyle necessitates the utilization of a definite technique with meticulous attention to details.

Because of the anatomic relation of the condyle to the skull the possibility of throwing this part of the mandible into relief on the roentgenogram has not always been easy of attainment. Various techniques have been advocated. The use of the same procedure as in making a lateral jaw exposure, accentuating the obtuseness of the angle formed by the central rays and the cassette, is not wholly satisfactory. If the condylar process is shown, invariably there is a disproportionateness of the jaw. When a complete survey of the mandible is made in one exposure, the existence of fractures of the condyle is usually established; however, accuracy as to the location of the fracture line is impossible of determination. In consideration of this remissness we have resorted to other methods which have proved in our experience more adequate and dependable.

* Instructor, Harvard University Dental School.

TECHNIQUE

Whether or not injury has involved the condylar head may be determined by making an exposure as for the mastoid region. The tube is placed immediately above the external auditory meatus of the opposite

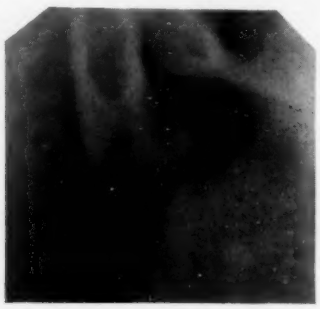


Fig. 1. Fracture of neck of condyle. Central rays passed through opposite sigmoid notch.

side. The rays pass through the temporal bone and strike the cassette, which is parallel to the side of the head, at an angle of 45°. Thus a good view of the temporomandibular articulation is obtained. Since this exposure is made with the mouth partly open, the failure of the condyle to move from the position occupied by it during closure of the jaws constitutes definite diagnostic evidence of fracture. The other method is used to determine the extent of injury to the neck of the condyle. The cassette is held parallel to, and against the side of, the face. The central rays are passed through the opposite sigmoid notch and directed at right angles to the condylar process. A slight deviation of the patient's chin toward the tube is desirable in order to throw the process into the path of the rays.

When consideration is given to the anatomic relation of the zygomatic arch to the skull, it is not surprising that roentgenograms of the arch or of the malar bone would very often prove unsatisfactory. The natural bulge of the skull in the region of the temporal and frontal bones casts shadows which obscure the zygomatic region. A roentgenogram taken vertically from below upward (Stone's position)[3] tends to throw both arches into relief. This method is suitable only for obtaining a general view; details as to the nature and extent of the fracture are missing. Because of this insufficiency of roentgenographic information, efforts were made to find another more satisfactory technique. The method here presented and developed by one of us (F. R. McG.) has not, so far as we are aware, been previously published. That it has proved eminently satisfactory and has furnished the accurate details that were sought is evidenced by its very practical value in the diagnosis of fractures involving the zygomatic arch.

This method involves the use of an intraoral technique. An occlusal film held in a cassette is placed within the mouth with the emulsion side of the film toward the maxilla. The film is slid as far out in the cheek on the side of injury as is possible; it is then held in position by means of the patient's closing the teeth together. The tube is placed above the head which is tilted slightly toward the opposite side to prevent

Fig. 2. Fracture of zygomatic arch. Film held within mouth, rays passing from above at right angles.

obscuring of the arch by the convex portion of the frontal bone. The central rays are directed at right angles to the film upon which an accurate impression of the zygomatic arch is made.

SUMMARY

Because of the anatomic position, fractures of the condyle of the mandible and zygomatic arch are difficult to show on the roentgenogram. In this paper are described two older methods for distinguishing fractures of the condyle and a procedure for diagnosing fractures of the zygomatic arch, relative to which perusal of the literature has failed to disclose any previous mention.

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3. Gillies, H. D., Kilner, T. P., and Stone, D. Fractures of malar-zygomatic compound; with description of new x-ray position. *Brit. J. Surg.*, 1927, *14*, 651–656.